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Lectures on diseases of the nervous system



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LECTURES

ON

DISEASES OF THE NERVOUS SYSTEM.

BY

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PROFESSOR OF PSYCHOLOGICAL MEDICINE AND DISEASES OF THE NERVOUS SYSTEM, AND OF MEDICAL JURISPRUDENCE, IN THE MISSOURI MEDICAL COLLEGE; PHYSICIAN TO ST. VINCENT'S INSTITUTION FOR THE INSANE; LATE SUPERINTENDENT OF THE ST. LOUIS COUNTY INSANE ASYLUM; CORRESPONDING MEMBER OF THE NEW YORK SOCIETY OF NEUROLOGY AND ELECTROLOGY; MEMBER OF THE AMERICAN NEUROLOGICAL ASSOCIATION.
ETC.

REPORTED BY V. BIART, M.D., REVISED AND EDITED BY
THE AUTHOR.

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TO

L. CH. BOISLINIÈRE, M.D.,

PROFESSOR OF MIDWIFERY IN THE ST. LOUIS MEDICAL COLLEGE,

THE ACCOMPLISHED OBSTETRICIAN,

CHRISTIAN GENTLEMAN,

AND UNSELFISH FRIEND OF THE YOUNG MEN OF THE MEDICAL PROFESSION,

This Volume is respectfully dedicated,

AS AN EVIDENCE OF AFFECTIONATE REGARD,

BY THE AUTHOR.

41127

P R E F A C E.

THIS course of lectures, delivered during the winter of 1874-75, was not originally intended for publication; but, at the urgent and repeated request of many of the alumni of the Missouri Medical College, the author has devoted all possible moments of leisure, whilst not engaged in his professional duties, to revising, correcting, and preparing for the press the copious notes taken by V. Biart, M.D., his former pupil.

In offering this volume to the profession, the author cannot, as an inducement to its perusal, claim for it originality either as to its facts or as to its theories. It is particularly designed for students and practitioners who have neither the time nor the opportunity for recourse to numerous authorities,—the aim being to present a thorough digest of the extensive field of medical literature on the subject of nervous diseases, and at the same time, so far as is consistent with a true portraiture of the maladies delineated and a clear idea of their characteristic features, to avoid diffuseness of detail and of description.

The author feels it incumbent upon him to express his grateful acknowledgments to the following writers, from whose works he has freely quoted, viz., Elam, Storer, Carpenter, Morel, Niemeyer, Jaccoud, J. Lewis Smith, West, Russell Reynolds, Hammond, Maudsley, Blandford, Schroeder van der Kolk, Sheppard, Todd, Tanner, Aitken, Flint, Hughlings Jackson, Da Costa, Ray, J. P. Gray, Echeverria, Clymer, Anstie, Buzzard, Trousseau, S. Weir

Mitchell, Moorehouse, Keen, Brown-Séguard, Esquirol, Winslow, Bucknell, and Tuke.

In the lectures upon hyperæmia, partial cerebral anæmia, and general cerebral anæmia, too much credit cannot be given to Niemeyer and Jaccoud, whose works principally furnished their foundation. Upon insanity, the views of Maudsley, Van der Kolk, Blandford, and Gray of Utica, will be frequently recognized by those familiar with their writings. The classic representations and literary photographs of disease by the immortal Trousseau, the forcible diagnostic elucidations of Da Costa, and the beautiful and interesting theories of the late Dr. Bentley Todd, are constantly referred to in many of these lectures. The discourses upon spinal disease are replete with the results of the recent labors of Dr. Brown-Séguard. The descriptions of aphasia and locomotor ataxia have, to a great extent, been borrowed from the valuable systematic work of Hammond on Nervous Diseases, the author having adopted, without reserve, the pathological explanations furnished by this distinguished neuropathologist (who has so largely contributed to the American literature of the subject), as more satisfactory than any other with which he is familiar.

In conclusion, the author must in justice express his sincere thanks to Dr. Wm. B. Hazard, of this city, whose great research and well-known qualifications have been of invaluable assistance to the former in the execution of his task.

If students are enabled to derive information from which they have been precluded by the multiform labors of a college session, if hitherto obscure or overlooked points of value are rendered clear and satisfactory to them, the author will feel amply repaid for the time and labor thus expended in their behalf.

2106 CLARK AVENUE, ST. LOUIS, September, 1875.

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LECTURES

ON

DISEASES OF THE NERVOUS SYSTEM.

LECTURE I.

THE CEREBRAL CIRCULATION.

GENTLEMEN,—Under the influence of a variety of causes, a change in the quantity of blood in the brain occasionally takes place.

There are three membranes enveloping the cerebral substance, the outermost being the dura mater, certain folds or prolongations of which constitute the so-called cerebral sinuses; these are lined by the continuation of the serous membrane of the veins, and carry the venous blood from the brain. The innermost membrane is the pia mater, and it is from this source that most of the arterial supply finds its way into the substance of the brain, by means of the capillary vessels. The intermediate membrane is the arachnoid, which is reflected upon itself, the space between the folds constituting its cavity. This latter membrane does not, like the pia mater, follow the surface of the cerebrum into its depressions or sulci; on the contrary, it stretches over the depressions, and leaves between itself and the pia mater a space called the sub-arachnoid, in which is contained a liquid,—the cerebro-spinal fluid. The amount of this fluid in the cranial cavity is not constant, but is in inverse ratio to the amount of blood in the vessels.

Some have maintained that the brain, being tightly held in the closed cranial cavity, will not admit of any variation in the quantity of the blood therein contained unless there should occur an inverse

variation in the quantity of its solid contents. In contradiction to this opinion, the theory has been advanced that a closed cavity is not absolutely present, and that the quantity of blood circulating in the brain may be augmented by cerebral compression, the result of which would necessarily afford increased capacity with corresponding reduction of volume.

Were the cerebral substance strictly compressible, the amount of cerebro-spinal fluid might actually remain constant; for when a larger amount of blood than usual found its way into the brain, its solid constituents would simply be forced to occupy a smaller space.

This, however, is not the fact; and although, perhaps, by pressure, you might bring the several particles of the brain into closer proximity, you could not, without causing molecular disintegration, make them occupy less space than previously.

The brain may be compared, as was cleverly suggested by Dr. Elam, to a sponge filled with fluid. By firm pressure you can cause all of the latter to escape from the porosities of the former. You may make the sponge apparently smaller than it was before the fluid was expressed, but it is impossible to reduce absolutely its volume by any pressure you may exert. The pores which existed in its substance, it is true, have been obliterated; but the constituent particles of the solid have by no means been compressed into a smaller space, which could only be accomplished by their actual encroachment each upon the other,—an occurrence which, if possible, would doubtless lead to their ultimate disorganization. Therefore Dr. Elam clearly shows by his argument that the human brain is certainly incompressible. Dynamically speaking, however, a pressure may be exerted materially influencing this supreme nerve-centre,—not by a reduction of its size, but by producing unmistakable effects upon its molecular structure, through the active circulation of blood when flowing in undue quantities, and under a variety of pathological conditions, in its delicate and highly-organized substance.

Hence vascular tension, occasioned by fluctuations in the quantity of the cerebral circulation, becomes an important factor in the explanation of many of the phenomena which we have to study.

We can readily understand that fluxion and stasis may eventuate

in molecular changes,—profoundly influencing the interstitial nutrition of an organ, so remarkably sensitive and impressionable, in consequence of the activity of its physiological tissue-metamorphoses. This blood-pressure may be better understood if we consider Dr. Elam's illustration, which most forcibly struck me in reading his valuable book. He states that "glass is sufficiently incompressible to be considered altogether so; yet glass may be subjected to pressure; and the effects upon its molecular structure are very striking and suggestive. Even the slight pressure that may be exerted by the fingers is sufficient very materially to alter its optical properties, especially as to its relation to polarized light; and the change continues so long as the pressure is continued."

It may, in conclusion, be safely affirmed that whilst the quantity of *blood* circulating in the cranial cavity may undoubtedly vary, the quantity of *liquid* there present is constantly and uniformly the same; which fact we have already stated is explained by the inverse ratio of compensation existing between the vital current and the cerebro-spinal fluid.

Congestion plays a most important *rôle* in the study of all conditions in which as a *primary* factor—whether active or passive—it produces serious disturbances, with an accompanying train of morbid phenomena. *Secondary* congestions—a result of a multitude of varying pathological cerebral lesions—are of no less interest, and offer the only reasonable interpretation of numerous fluctuating symptoms and daily mutations in the condition of patients affected with chronic cerebral disease. This peculiar variation of symptoms,—the daily alternation of those of excitation with those of depression,—the appearance and disappearance of the disturbances of motility within a few hours in certain cases of softening, may be thus explained satisfactorily. Before proceeding further with this subject, since during this course of lectures constant reference must be made to the different portions of the encephalon and its physiological functions, it might be judicious to make some general remarks regarding our ideas of the natural anatomical divisions of this great and truly wonderful accumulation and arrangement of ganglionic cells or batteries. Jaccoud's description is the one we have adopted, because it recommends its claims by challenging our admiration and preference in consequence of its beautiful simplicity

and apparent accuracy,—coinciding as it does with the advanced physiological discoveries of the present day, and marvelously corresponding with the opinions expounded by Dr. Maudsley in his great work on the “Physiology and Pathology of the Mind.”

According to Jaccoud, the nervous system is divided into three distinct apparatuses:

I. The spinal apparatus, comprising a peripheral portion (the nerves); a rachidian portion (the spinal marrow); a cephalic portion (medulla oblongata, tubercula quadrigemina, cerebral peduncles, and pons varolii), to which is annexed the cerebellum.

II. The cerebral apparatus, composed of the cerebral hemispheres exclusively.

III. An apparatus of conjunction interposed between the two anatomical divisions just described, and composed of symmetrically situated dual organs,—the thalami nervorum opticorum, and the corpora striata.

The connection of the cerebral and spinal apparatuses is direct, and accomplished by the intervention of the gray substance of the optic thalami and corpora striata,—which is the receptacle and point of departure of the white fibres of the two varieties of apparatuses which are in communication.

The anatomical facts justifying this division are as follow: the fibres of the cerebral hemispheres, and those belonging to the spinal apparatus, are independent, distinct, and individual; the cerebral fibres emanating from the gray superficial or peripheral zone of the hemispheres do not pass beyond the corpora striata and the optic thalami; in these intermediate organs, by means of the gray substance, they enter into relation with the terminal fibres of the inferior or spinal apparatus; in conclusion, the cerebral apparatus gives off no nerve,—all the peripheral conductors springing from the spinal apparatus. The physiological facts indicate more plainly still the truth of this division, because they place in remarkable contrast the respective rôles of the three secondary systems. General physiology teaches that the vital activity of man admits of three distinctions or classifications: *vegetative*, *animal*, and *intellectual* life. *Vegetative activity*, inasmuch as it depends upon nerve-centres, is influenced only by the spinal apparatus; *intellectual activity* can originate from the cerebral apparatus alone; whilst *animal activity* is common to both. In this common sphere

of action, however, we can seize a distinctive character of the utmost importance: the mere animal acts are voluntary or involuntary; those of pure volition belong exclusively to the cerebral, whilst the automatic or involuntary spring entirely from the spinal apparatus. So far I have literally transcribed the words of Jaccoud, as a more faithful portraiture of his views than any abstract I might offer. Their importance will be conceded by all interested in this subject, and our recurrence to them will be frequent, inasmuch as our adoption of his classification is without qualification.

To return to the subject of this lecture, from which we have intentionally made this long digression, we may unequivocally maintain that in order to change the relative amount of cerebro-spinal fluid we must have an increased quantity of blood in the brain. This *quantity*, however, is very variable, as is also the *quality* of the blood which is in excess.

These quantitative differences are embraced under the one collective and generic term, hyperæmia of the brain.

LECTURE II.

HYPERÆMIA OF THE BRAIN.

Definition.—Active Hyperæmia.—Causes: Emotions, Fevers, Diseases, Slight Resistance of Capillaries, Pressure, Malaria, Cold, Atrophy of the Brain, Paralysis of Vaso-Motor Nerves, Alcohol, etc.—Irritation of Vaso-Motor Nerves: Poisons, Alcohol, Excessive Mental Work.—Passive Hyperæmia.—Causes: Strangulation, Pressure, Expiratory Efforts, Impediments to the Heart's Action, Compensation, Altered Structure or Function of the Lung.—Post-Mortem Changes.—Active Congestion.—Anæmia produced by Collateral Œdema.—Forms of Hyperæmia.—Symptoms of Mild Hyperæmia.—Severe Form.—Delirium.—Insanity.—Hallucination.—Illusion.—Apoplectic Form.—Common Symptoms.—Diagnosis.—Prognosis.—Treatment.

GENTLEMEN,—By hyperæmia is meant an undue or excessive determination of blood to a part.

Hyperæmia of the brain is generally divided into two kinds: *active* and *passive*.

By *active* hyperæmia, we mean arterial or acute fluxionary congestion. By *passive* hyperæmia, we understand venous or congestive hyperæmia.

The causes of hyperæmia are numerous, and among them we find—

The *emotions*. We have all experienced the rush of blood to the head, preceded by violent throbbing of the heart, and followed by heat in the upper portion of the body, caused by violent mental emotions. Ordinarily these emotions are not of a grave nature, and consequently their effects are transitory. It happens, however, that their violence may be such that the hyperæmia may result in severe, nay, fatal consequences. Hyperæmia in this case is due in part to increased cardiac action and partly to paralysis of the vaso-motor nerves.

Another frequent cause of hyperæmia is *fever*, which produces this morbid condition by increased cardiac action, blood-poisoning, and excessive elevation of temperature.

Certain pathological conditions produce it, as hypertrophy of

the left ventricle of the heart, which is sometimes present in Bright's disease of the kidneys; or non-complicated and non-compensatory hypertrophy.

A not infrequent cause of hyperæmia is too slight amount of resistance of the capillaries, in consequence of certain peculiarities in their structural conformation.

Undoubtedly one of the most direct causes of hyperæmia is pressure by tumors upon certain portions of the aorta. Most of the blood supplying the brain being carried there by the carotid arteries, if in any way a pressure upon the thoracic or abdominal part of the descending aorta should be exercised, it is evident that the greater volume of this blood must be carried upwards, and consequently to the brain, thus giving to this organ an undue supply.

In some conditions of malarial poisoning we have hyperæmia during the algid stage, produced by diversion of the blood from the peripheral cutaneous vessels to the more deeply-situated organs. It frequently happens that extreme cold, in this manner, becomes a grave cause of hyperæmia. The explanation in this case is extremely simple. We all know that cold contracts the capillaries, and consequently produces an increased current to the internal parts. A great deal of this blood goes to the brain, and, as it is arterialized, it irritates the nervous system and produces symptoms of congestion. As mentioned by Watson, men perfectly sober have been arrested in the streets during very cold weather for being drunk, when the cause of their strange behavior was traced to the effects of the extreme cold.

Insolation, sudden arrest of hemorrhoidal or menstrual discharges, night vigils, and excessive indulgences in the pleasures of the table, are all prolific sources of dangerous attacks of active cerebral congestion. Atrophy of the brain is still another source of hyperæmia. Atrophy of the brain itself may occur in many ways; but when it exists, and a portion of the brain has wasted away, a vacuum forms,—or rather would form, were it not that, in consequence of a compensatory vascular dilatation, the wasted brain is replaced by blood; and thus we have here also another cause of undue determination of blood to that organ.

One more cause of hyperæmia, the result of a diversity of influentes, is yet to be enumerated. I allude to paralysis of the vaso-motor nerves, accomplished in many and different ways,

prominent among which are: section of certain nerves, emotions, excessive intellectual labor, narcotic poisons, and, very frequently, abuse of alcoholic stimulants. In order to understand fully the manner in which paralysis of the vaso-motor nerves causes hyperæmia, we must, evidently, first consider the functions of these nerves, the neuro-physiology of the vessels to which they are distributed, and the presiding influence exerted by these nerves over their proper innervation. The blood-vessels are furnished with a certain elastic coat, which, by alternate expansion and contraction, regulates the flow, and consequently the supply, of blood to certain parts. As the stimulus to the organs of the body is derived from nervous centres, and conducted by nerves, it follows that this contractile coat must also be furnished with a nerve, which in this case is derived from the great *sympathetic*, from which all vaso-motor nerves emanate.

The nerves subserving this function are called the vaso-motor nerves,—in other words, the nerves regulating the movements of the vessel. When no pernicious influence acts upon these nerves, they will normally perform their functions, unless the coats of the vessels themselves have undergone some change in structure, when it would be impossible for them to respond to the directions of the nerve-force. But where injurious impressions are exerted, they seriously interfere with the appropriate action of the nerve, and more or less impede or augment the circulation. When a vaso-motor centre or its nerve is irritated, the arterioles thereby supplied immediately contract. But when a paralysis of these nerves exists, the reverse takes place: the nerves lose their excitability and are unable to transmit the command for contraction, the arterioles remain as a consequence dilated, and hyperæmia results.

It has already been observed that the causes of this paralysis are various, those most prevalent being such poisons as opium, belladonna, hyoscyamus, and many other narcotics. Alcohol is the most common source of mischief in this respect. Excessive intellectual work is also mentioned by some writers as a prolific and disastrous cause of hyperæmia.

Having heretofore considered the different causes of hyperæmia of an active character, it behooves us to review some of the causes of passive, venous, or congestive hyperæmia.

Passive hyperæmia, it is well to recollect, may be produced by strangulation, pressure on venous trunks, violent expiratory efforts, impediments to the functions of the heart, and altered or pathological conditions of the lungs.

There is nothing which exercises so much power in producing passive hyperæmia of the brain as strangulation: this is best exemplified in hanging, where congestion immediately takes place. The *modus operandi* is, that it simply opposes or prevents the return of venous blood to the right side of the heart by a mechanical obstacle. Pressure upon venous trunks produces results identical with those of strangulation, by obstructing the return of venous blood to the heart. Tumors in the neck pressing upon the jugular vein, or aneurism in the thorax pressing upon the vena cava descendens, may be cited as causes having a similar result. The violent expiratory efforts, before alluded to, are often witnessed in persons playing upon wind-instruments requiring the forcible expulsion of air from the lungs. Loud and prolonged singing and speaking, the straining of parturient women, and violent muscular exercise, may be classed in the same category of causes. A better illustration is afforded by the rapid succession of expiratory acts in whooping-cough.

The impediments to the heart's function are numerous, and generally productive of hyperæmia of a congestive form. Let us suppose, by way of illustration, that we have a case of disease of the aortic valves, accompanied by regurgitation. How will this produce hyperæmia? The arterial blood has not perfect exit from the left ventricle of the heart during the systolic contraction, and hence prevents the free entrance of the blood, through the mitral valves, returning from the lungs; the natural result is that the arterial blood, being unduly retained in the lungs, obstructs the venous blood flowing from the right ventricle, the obstruction in the right ventricle is soon appreciated by the right auricle, and finally, the free entrance of blood from the vena cava descendens being obstructed, we will have passive congestion of the brain. There is in nature, however, a grand force which exerts itself whenever occasion demands,—the principle of compensation. In obedience to this salutary law, one organ being diseased, another performs double labor; or one part of an organ being injured in any way, the healthy portion makes up for the deficiency by increased

action. These compensations will necessitate an hypertrophy, which generally occurs, especially in cardiac lesions of a valvular nature. The left ventricle hypertrophies and performs increased duty, expelling the blood from its cavity with augmented force. When the compensatory action is sufficient, there will be no congestive hyperæmia resulting.

The last cause we have to consider is the alteration in the physiological functions in the lungs, with or without change of structure, resulting in simple capillary obstruction. In hydrothorax, and emphysema of the lung, there is in the former case a liquid effusion, and in the latter a larger volume of air, pressing upon the capillaries, obstructing the pulmonary circulation, and impeding the flow of blood from the right ventricle. The right auricle becomes engorged with blood, a large column of which fills the jugular veins, engendering passive hyperæmia of the brain. It must be observed that in this case, as well as in the other, nature oftentimes endeavors to compensate for the lack of power of the right ventricle to propel the blood through the obstructed capillaries, by occasioning hypertrophic changes in its walls.

It is not always easy to determine, upon autopsy, the previous existence of hyperæmia. The effects of passive hyperæmia are quite different from those of active hyperæmia, and not very liable to be confounded with them. In *active hyperæmia* some of the principal results are great vascularity of the affected part, sanguinolent succulence, as evinced by numerous *puncta vasculosa*, and the exudation of serum, occasioning œdematous infiltration. In *passive hyperæmia* (congestive) we may find the brain hyperæmic, with a venous plethora, at the expense of an arterial anæmia. However, neither of the conditions above described is conclusive of the cause, which may depend upon many different circumstances. Indeed, in post-mortem examinations, incorrect deductions as to the existence of hyperæmia are very apt to be made, the arteries at the base of the brain being naturally large, and oftentimes their post-mortem fullness is undoubtedly influenced by the tendency on the part of the blood to gravitate. The distended and tortuous blood-vessels on the top of the brain are venous, and quite capacious on account of their size. These vessels are almost always found empty after a long-continued and exhausting disease. The vessels outside of the brain, therefore, afford no definite evidence

of hyperæmia, nor does the presence of a large or small quantity of blood in the substance always give us any positive information on which to build our conclusions for the supposed presence of ante-mortem congestion.

Since the brain-substance receives blood from capillaries proceeding from the pia mater, it must be evident that the vessels in the cerebral substance proper are too minute to be seen by the naked eye. We can, it is true, judge of the quantity of blood these capillaries contain, approximately, by making a transverse section of the brain, causing them to become apparent to us as little dots, called the *puncta vasculosa*. But even these are not thoroughly significant, for the rapidity or freedom with which the blood exudes from them is often due to change in its constitution. When the blood runs slowly, it is often due to a state of hyperinosis; when freely, it is often owing to a deficiency of fibrin in it, which frequently happens in cases of dyscrasia. Here, once more, we have no infallible test for determining the presence or absence of hyperæmia which had previously existed. The knowledge of the effects of hyperæmia will undoubtedly throw much light upon our investigations, and enable us to recognize many pathological conditions which would otherwise escape our observation.

In cases of active congestion, the capillaries are distended, the blood moves very slowly through them, and perhaps also, in consequence of some cause or other exercising constantly-increasing tension on the coats of the vessels, there is developed upon the part of the capillaries a proneness to transude serum. When the congestion continues for some time, this last result is undoubtedly the danger to be feared. This serous transudation is known as

COLLATERAL ŒDEMA.

It is evident that the collateral œdema cannot distend the membranes, though it nevertheless exerts a constant pressure. Does it compress the brain? Of course not. The fact of the brain's incompressibility has already been established. What, then, sustains the constraining impulse? It compresses the capillaries, the very vessels where it originated. These vessels being the only elastic portion of the brain, and having the pressure of the collateral œdema exercised upon them, their calibre is diminished or entirely

obstructed, and a resulting anæmia is produced. In other words, the parent vessels are strangulated by their own progeny,—the collateral œdema. You must not suppose, however, that collateral œdema necessarily occurs in all cases of congestion. Its amount is proportional to the intensity of the congestion. In death from congestive hyperæmia, therefore, we generally find the affected districts more or less anæmic.

There are three forms of hyperæmia, the mild, the severe, and the apoplectic. A few symptoms are common to all forms of hyperæmia. Those generally found in the mild form are:

1. Increased excitability and general hyperæsthesia of the nerves of special sense.

2. Contraction of the pupils.

3. Insomnia, or vivid and frightful dreams.

4. Vomiting.

5. A flushed face; which, however, is sometimes absent.

6. A torpid condition of the bowels.

7. Headache, sometimes violent.

8. A constant buzzing in the ears.

9. Dizziness in proportion to the severity of the attack.

10. Motor and sensory symptoms of excitation, more or less marked.

11. Light and noises are badly tolerated.

12. More or less psychical disturbance.

In the severe form, characterized by great intellectual excitement and disorder, nearly all of the above symptoms present themselves, increased in intensity and accompanied by delirium, hallucinations, illusions, and delusions. Simple delirium is a wandering of the mind, oftentimes attended by fever, of which it may be the result. It is not a symptom, necessarily, of hyperæmia; but rather points to blood-poisoning, or excessive tissue-oxidation, accompanied by increased elevation of temperature.

A man is, in a certain sense, insane when he is unable to use the mental powers necessary to dispel hallucinations, which are nothing but conceptions of things having no existence outside of the patient's brain. If he imagines wild animals jumping about upon his bed when he is alone in his room, he labors under an hallucination; while if he perverts the impression received in his brain from external, sensible objects, as, for instance, if he

imagines a chair to be a living object, approaching or threatening, he then has illusions.

Delirium tremens should never be confounded with ordinary hyperæmic delirium, from the easily-recognized fact of the involuntary tremor, coinciding with a characteristic good-natured and loquacious delirium, almost pathognomonic of the former.

The word apoplexy is generally used to express the effects produced by an extravasation of blood into the cerebral substance. By the apoplectic form of hyperæmia, however, is meant that variety whose effects are somewhat similar, so far as symptoms go, to those of apoplectic extravasation. The main symptoms of this form are sudden loss of consciousness, and abolition of sensation and voluntary motion.

The different symptoms enumerated as belonging to the several forms of hyperæmia of the brain are also common to other affections, and it is essential to be able to discriminate between them, in order not to adopt a treatment based upon an incorrect diagnosis, which in certain instances might prove dangerous, or even fatal. Were we, for instance, to treat a hyperæmic patient for anæmia, we should be likely soon to sign his burial certificate, though the pallor sometimes present in hyperæmia might lead us to think the patient suffering from anæmia.

It happens that hyperæmia in children exhibits very severe symptoms, which may very closely resemble those of meningitis. The latter disease is very fatal, while the former is not necessarily so; and it therefore behooves us to guard against the error of imagining that we have controlled a meningitis, when in reality it never existed. The previous history will throw considerable light upon the case, and greatly aid in the diagnosis. If the child has been very well until the day preceding the attack, if it has suffered from no contusion about the head, or other severe injury, we may after a short lapse of time generally give a favorable prognosis, especially if the convulsions do not recur with frequency and the temperature be normal.

The symptoms of apoplexy may be induced by blood-poisoning (uræmia), which we must not confound with the apoplectic form of cerebral congestion. *Insolatio* (sun-stroke), which also destroys life by suspending the nervous energy, is not the result of hyperæmia, but, it is generally conceded, can be traced to an elevation

of the temperature of the whole body, so high as to be incompatible with the functions of life, and fatal to the proper performance of the duties of the nerve-centres. Stomachic vertigo presents symptoms congestive in character, but we should remember that it is never accompanied by loss of consciousness, and the symptoms disappear generally after the action of antidyspeptics to remove the cause. In congestion the *temperature* is not elevated, which makes it easy to determine whether or not the symptoms be due to fever. All we have to do is to place the thermometer in the axilla, and the diagnosis is rendered certain. The symptoms are always of *short duration*; a point of the greatest importance and significance, as a prolongation of the symptomatic indications would cause grave suspicions of serious lesions.

One marked characteristic feature of the symptoms of cerebral congestion is, that they are *general* and diffused, not localized or limited.

In congestion the breathing is regular, not stertorous, and the pulse is but little accelerated, though usually quite strong. In syncope, on the other hand, the breathing is impeded, the pulse very feeble and irregular, and the face *remarkably* pale. This last fact must not have undue importance attached to it, since we know that in hyperæmia of the most dangerous type the countenance is sometimes cadaverous.

Loss of consciousness being characteristic of cerebral hemorrhage, epilepsy, and other comatose conditions, we may be at a loss to determine its cause. If in such a dilemma we wait until the ordinary period for an epileptic fit to pass off, and by differential diagnosis exclude other apoplectic states, we can soon discriminate between the presence and the absence of congestion. The phenomena attending apoplectic hyperæmia are transient; in epilepsy they last but a few minutes; and in apoplexy, if the coma be not fatal, they may last for days.

An epileptic attack is oftentimes accompanied by convulsions. Immediately examine the tongue of the patient: you will frequently, though not always, find it to be lacerated. The control of the sphincter muscles, in this convulsive disease, is generally lost, and you will find, as a frequent consequence, an involuntary discharge of fæces and urine.

An examination of the abdomen and thorax may lead to the

discovery of an aneurism or tumor pressing upon some important blood-vessel, or enable us to detect some cardiac or pulmonary lesion, causing determination of blood to the head.

As a slight evidence of the facility with which a mistake may be made in the diagnosis and prognosis of cerebral hyperæmia, I will recall a case of more than usual interest, which indelibly impressed upon my mind the necessity for caution in this respect. A very distinguished medical man, about fifty years of age, whose life-long habits of study and excessive intellectual labor had brought on serious brain-symptoms, was placed under my charge. His physicians, who were eminent practitioners, had diagnosed cerebral softening, the result of thrombosis. Upon examination, the patient was found to be perfectly incoherent; his delusions were marked and dangerous in character; his countenance indicated a hebetude amounting almost to imbecility; aphasia was a prominent symptom, and agraphia very pronounced. After desperate efforts to write, he folded with care a paper on which were traced a few illegible hieroglyphics, and, placing it in an envelope with *no address*, handed it to me with a request that I should deliver it to a relative whom he named. Upon my calling his attention to the fact that the name and address had been omitted, he became quite excited and irritated, and insisted that I could not read. Amnesia to a limited extent existed, but it was to me a fact of great significance that, notwithstanding his utter deficiency of normal ideation and the presence of other grave and alarming symptoms, his memory of the past and his interest in the present were far from being greatly impaired. My observation and experience having tended to make me believe that amnesia and decided apathy are the ever-present, concomitant, and characteristic symptoms of cerebral softening, I made a particular note of their partial absence in this case. The patient also presented some sensory and motor disturbances: he was partially hemiplegic on the right side, and the orbicularis oris was implicated, as was evinced in a depression of the right labial commissure, which permitted the saliva to escape and dribble down his face, greatly adding to the stolidity of his appearance. His eyes were injected, and his emotional faculties were preternaturally mobile, as was made apparent by his alternate attacks of weeping and laughing. I was made conversant with the fact that the patient had for

several years greatly abused the use of narcotics and stimulants while seeking relief from terrible attacks of neuralgia, to which he had been a victim all his life. This indulgence, as usual, was due to the injudicious advice of various physicians. Medical men are oftentimes not a little to blame for the moral, physical, and intellectual wrecks occasioned by their countenancing an imprudent resort in their patients to these dangerous and potent remedies with a view of relieving temporary pain. They seem to ignore the fire they not infrequently kindle, by the creation of a morbid appetite at times so difficult to restrain, too often, alas! impossible to appease. The patient had a puffed, bloated appearance, which to an experienced eye strongly indicated the familiar evidences of chronic alcoholism. His father had died insane, and many members of his family were distinguished for their vagaries and eccentricities, probably being the possessors of the "neurosis spasmodica," living as it were upon the border-land of insanity. The outlook of this case, therefore, was certainly dark; yet upon a careful investigation and analysis of all the symptoms, and particularly laying stress upon the history of the disease, which was that of alcoholism, I diagnosticated cerebral congestion, the result of vaso-motor paralysis induced by his indiscretions, for which, however, I believe he was in no degree morally culpable. Basing the treatment upon the conclusions I had ventured to adopt, I gave him a preparation containing full doses of Squibb's fluid extract of ergot, digitalis, and muriated tincture of iron, to be taken three or four times daily; regulated his constipation with aloetic laxatives, obviated his insomnia with a combination of bromide of potassium and hydrate of chloral, and carefully made him eschew all malt, vinous, and alcoholic stimulants, together with his favorite narcotics.

Had the diagnosis of his previous attendants been correct, such a therapeutic course would have been unjustifiable, not to say destructive. I had the gratification to see him rapidly recover, and in two weeks nearly all his symptoms disappeared, and in two months he was discharged cured. The hyperæmia in his case was doubtless excessive, and the resulting collateral œdema must have been considerable, and a nice point was to ascertain whether the presence of the latter in the delicate brain-substance might not have wrought some disastrous structural changes, oc-

casioned by capillary anæmia inducing nutritive perversion, in which case the damage would have been irretrievable even after the cessation or disappearance of the primary congestion. Such, however, was not the case, the patient's restoration being complete. It therefore behooves you, gentlemen, to bear in recollection and appreciate the fact that without a proper medical *history* our best efforts at diagnosis will oftentimes be rendered futile.

PROGNOSIS OF HYPERÆMIA.

One attack of hyperæmia predisposes to another, and the repetition of them may result in atrophy, softening, or some other serious lesion of the brain, resulting from the profound nutritive derangement generated by the dynamic influences of these repeated fluxions.

A tendency to congestion in other organs, especially the lungs, is a contingency not infrequent in occurrence, and one which we should anticipate.

The hyperæmia induced by the intemperate use of alcohol is sometimes tenacious, while that caused by anxiety or excessive mental labor, or by the suppression of natural discharges, is generally relieved by the removal of the cause.

TREATMENT.

Whenever it is practicable, the first step in the inauguration of a successful plan of treatment is to ascertain and remove the cause of the congestion. The application of cold to the head is most generally advisable. A very eligible mode of applying it is to introduce pounded ice into bladders or rubber bags and lay them on the patient's head.

Purgatives constitute most efficient therapeutic measures, producing marked derivative effects. Among the many that can be recommended, a combination of jalap and calomel (about ten grains each) deserves especial notice.

The local abstraction of blood is often resorted to beneficially in cases of hyperæmia resulting from suppression of certain discharges. It should generally be done at a distance from the brain, by the application of leeches to the pituitary membrane or the margin of the anus. The abstraction of blood from the general circulation, i.e., from the blood-vessels, is undoubtedly the

most necessary of all the means of treating certain varieties of congestion of the brain, due to intense collateral hyperæmia, or to increased pressure in the carotids, as a consequence of obstructed escape of blood from the aorta, and, lastly, in very threatening cases, where it would be dangerous to await the action of milder measures. Venous congestion, with few exceptions, requires bleeding of either a local or a general character. Hyperæmia from increased cardiac action, uncomplicated by valvular lesions, with undue accumulation of blood in the carotid arteries, imperatively demands such a course. In aneurism producing hyperæmia, or in cases of serious collateral cedema, bleeding should always be resorted to, keeping in mind the indications and urgency of the symptoms. It should not be essayed in cases of valvular diseases of the heart, or paralysis of the vaso-motor nerves, resulting from excessive mental efforts or from narcotic or alcoholic indulgences. These latter require energetic treatment without bleeding, and, in addition to the methods already given, we may resort to warm sinapisms. This last method may often be advantageously employed, the irritating effects produced by rubefacients and vesicants causing a derivative current of blood to the skin and other parts. *Ubi irritatio, ibi affluxus est.* Therefore the vesicants and irritants are most advantageously applied to the lower extremities.

We should not overlook the important fact that delirium is increased by temperature. The reduction of the temperature is often effectually accomplished by the judicious use of quinine and alcohol. This has reference, however, to *delirium produced by fever*, and not to that of congestion, as these remedies would, in the latter complication, prove most pernicious. There being no *elevation of temperature*, you have simply to deal with the *mechanical* effects of an undue determination of blood to the parts.

LECTURE III.

PARTIAL ANÆMIA OF THE BRAIN.

Definition.—Closure of Vessels.—Collateral Œdema.—Pressure upon Capillaries.—Thrombosis.—Embolism.—Rheumatism.—Thrombosis as a Cause of Embolism.—Aneurism as a Cause.—Artificial Production of Embolism.—Effects of Closure.—Collateral Circulation.—Ligation of Carotid in Man; in Animals.—Embolism in Left Side.—Why the *Right* Side is generally paralyzed.—Fissure of Sylvius.—Brain not Gangrenous.—Cause of Absence of Gangrene.—Cause of Presence of Gangrene.—Collateral Hyperæmia.—Secondary Anæmia.—Compression of Capillaries.—Change of Color.—Size of Softened Parts.—Anatomical Condition in Anæmia.—Pathological Effects of Pressure.—Symptoms of Softening of the Brain.—Degrees of Functional Derangement.—Symptoms of Excitation and of Depression: Amnesia, Agraphia, Aphasia, Hemiplegia.—Peripheral Arteries.—Variation of Symptoms.—Differentiation between Embolus and Cerebral Hemorrhage.—Differentiation between Thrombosis and Embolism.—Symptoms of Anæmia from Collateral Œdema.—Obscure Diseases explained by Collateral Œdema.—Explanation of Phenomena of Clot.—Symptoms of Pressure by Abscesses, Tumors, etc.—Obscurity of Diagnosis in Brain-Diseases.

GENTLEMEN,—Anæmia, as the term implies, is a deficiency of blood, as regards the quantity. It is, therefore, the opposite pathological condition to hyperæmia. Partial anæmia of the brain, according to Niemeyer's classification, is dependent upon several causes, that can be placed under three heads, as follows:

1. *Closure of the afferent blood-vessels* (by *afferent* vessels are meant those leading to a part, in contradistinction to *efferent*, those leading from a part). The anæmia from this cause may be general or partial: it is general when the entire encephalon is involved, and partial when limited to only one hemisphere, or to a portion thereof. In the majority of cases we have only a *partial* anæmia.

2. *Collateral œdema*. This is a transudation of the serum of the blood into the surrounding tissues. This serum presses upon the capillaries, and is the general result of active or passive hyperæmia. We have already noted its effects while reviewing hyperæmia, and it is evident that, in this connection, we might name hyperæmia as one of the causes of anæmia.

3. *Pressure upon the capillaries by tumors, abscesses, clots of*

blood, etc. In all instances we have softening of the brain as a direct result of a continued exclusion of blood therefrom. Let us consider each of these causes at length. First we have closure of the afferent blood-vessels. This is produced by two pathological conditions: embolism and thrombosis.

THROMBOSIS.

It happens in certain affections that the blood is not properly propelled through the vessels. This may occur in various ways. The arteries are all supplied with a fibro-muscular coat, controlled by the vaso-motor nerve accompanying the vessel. This insures the contraction and relaxation of the artery, which actions are both compensatory and essential to the proper and equal continuation of the circulation. Arterial elasticity and contractility may be lessened by the pressure of tumors upon the artery, or more particularly so by disease of the vessels themselves, as, for instance, in calcareous degeneration, or by inflammatory action, giving rise to a disease termed *endo-arteritis deformans*, which is more liable to attack aged people. Now, a result of this deficient propulsion of the blood is a retardation of the circulation, the inner surface of the arterial trunks being roughened, and a deposit of fibrin sooner or later occurs. This process is a slow one, and takes place also in the smaller arteries, thereby greatly impeding, if not preventing, the re-establishment of the collateral circulation. For it must be borne in mind that in *endo-arteritis deformans* the arterioles are in precisely the same condition, pathologically, as the trunks, and, in consequence of their non-dilatability, the collateral circulation is not affected. The clot or fibrinous deposit above described is called a thrombus, and always occurs *in situ*; that is, we find it where the pathological conditions above described are most active. In inflammatory affections the blood is hyperinotic, and consequently will more readily deposit its fibrin, and so in arteritis we find a concurrence of conditions very favorable for the production of thrombosis.

Between the clot of thrombosis (a thrombus) and that of embolism (an embolus) there need not be a great deal of difference. We have already seen that a thrombus obstructs the circulation at the place of its formation. When a clot obstructs a vessel at a point distant from the place of its formation, it is called an em-

bolus. The latter condition is characterized by suddenness of invasion, and the vessels whose walls confine or arrest it in its course may themselves be in an entirely normal condition. An embolus may originate from different organs of the body, the lungs, heart, etc., and it may be as varied in composition as are its points of departure. An embolus is generally conveyed from the heart, and is usually fibrinous in character.

Different abnormal states of the system may predispose to conditions of the cardiac valves and orifices which may subsequently induce embolism. Rheumatism is a very common cause. In this affection, due to a peculiar *materies morbi*, there is an inflammation of the several fibrous tissues of the body, and there is frequently developed a pericarditis, endocarditis, or thickening of the auriculo-ventricular and aortic valves. A deposit of fibrin follows, with the formation of concretions and vegetations, parts of which are perhaps detached by the current of the blood, carried away from the heart, and produce embolism in some distant artery. Or, if the original embolus is very friable, it may, as Virchow has shown, break up into minute fragments and constitute capillary emboli.

Thrombosis may sometimes be the cause of embolism, when, for instance, the thrombus is formed in a vein as a result of phlebitis, and a portion of its substance, becoming detached, is carried away in the venous current. When, on the contrary, it occurs in an artery, it closes the artery, and cannot be conveyed along by the current of blood, on account of the diameter of the vessel gradually diminishing. But the venous blood is conducted by vessels whose diameters *increase* in the direction of the current. Therefore, where a thrombus forms, as in phlebitis occurring in certain puerperal conditions (coagulation in the uterine sinuses, etc.), the clot, or a portion of it, becomes detached, and is carried along to the right auricle, right ventricle, pulmonary artery, and finally lodges, according to its size, in one of the larger or smaller pulmonary vessels. It is, therefore, evident that when a thrombus in a vein results in embolism, it will always be arrested in the lungs or in the trunk of the pulmonary artery.

As the main cause of thrombosis (*endo-arteritis deformans*) only attacks persons who have passed the meridian of life, it materially differs from embolism in this respect, that the latter is not con-

fined to any age, and may occur at any moment and under varying conditions.

Embolism, as has been previously stated, may be produced by a diversity of causes, though it generally originates in the heart. It has been known to result from the handling of an aneurism containing a clot of fibrin, where the clot or a portion of it became detached, and the patient perished from embolism. Physiological experiments have been made with the view of producing embolism artificially, and this has been done by the injection of solid foreign bodies, such as millet-seeds, etc., into the blood-vessels of animals, producing a closure of the afferent vessels, and resulting in disturbances of the circulation in the organs affected.

The next consideration will be some of the effects resulting from the closure of vessels supplying the cerebrum with blood. Suppose the left middle cerebral artery to be suddenly plugged, —and a very important one it is, supplying a part of the hemisphere with blood. The first result will be an anæmia of that portion of the hemisphere which cannot receive its blood-supply from the artery, now no longer pervious, and, if it be not relieved, that hemisphere will cease to perform its functions, and the condition persisting, there will be a lack of nutrition, and, as an inevitable consequence, softening.

We have said, when speaking of thrombosis, that in this morbid state the arterioles are in the same condition as the trunks, having lost their resiliency, which is so eminently necessary for the normal propulsion of the blood. It therefore follows that the collateral circulation cannot be established. How is it in embolism, where no pathological state of the collateral vessels exists? Farther on, it will be seen that it depends entirely upon the situation of the artery which lodges the embolus whether or not the collateral circulation will be developed. If the embolus be lodged in an artery *below* the circle of Willis, the collateral circulation quite possibly will occur; but such will not be the case if the plug or embolus be in an artery *above* the circle of Willis, and the portions of the brain deprived of their blood-supply will become anæmic, because the other vessels will not be adequate to send a sufficiency of blood to supply the territory deprived. Where rheumatism, for instance, is the predisposing cause, there is first an embolus producing

anæmia, and this condition will eventuate in softening, and lastly hemiplegia, with abolition of the psychical functions, more or less marked, according to the site of the pathological lesion. Whenever, therefore, the embolus lodges *above* the circle of Willis, there is no probability of recovery from the disease, though life may be prolonged. It is, for this reason, plainly a much more favorable condition for the clot to lodge *below* the circle of Willis; but even then the situation is sometimes grave, for in man there are difficulties opposing, or at least unfavorable to, the establishment of the collateral circulation, and softening very often results. In corroboration of this statement may be cited the well-known fact of softening sometimes ensuing upon ligation of one of the carotids. This should not deter the surgeon from ligating either of these arteries where necessity demands it. In certain animals the tendency always seems to be in favor of the re-establishment of the circulation, and experiments have been made upon rabbits, where no bad effects ensued from the ligation of three of the four supplying arteries. Of course, where the softening occurs on the left side of the brain, the hemiplegia will be upon the right side of the body; and you will find it to be a well-known clinical fact that the right side is generally the one paralyzed. The reason of this is simple. The right common carotid artery arises from the *arteria innominata*, while the left arises directly from the arch of the aorta, and is, therefore, more in the line of direction of the arterial current; hence it follows that an embolus from the heart will more readily enter the left than the right carotid. The embolus is, therefore, frequently arrested in the left middle cerebral artery, in the fissure of Sylvius. In autopsy, it is always prudent to examine this artery, especially when other anatomical explorations have offered only negative results.

Another point, which it is well to refer to, is that the brain does not become gangrenous, although Niemeyer speaks very truly of the condition present being one of necrosis. It softens, liquefies, becomes disorganized, but it does not putrefy or become fetid. The different influences (of atmosphere, temperature, etc.) which are requisite for such a condition are almost entirely excluded, the brain being closely confined and hermetically protected by the skull. Nevertheless, you may find offensive abscesses or putrid collections within the skull; but this happens only when the

embolus originated in a necrosed point, and hence carried the infection with it from the source whence it emanated. We may have such a condition of things in certain pulmonary affections; as in tuberculosis, where there is considerable tissue-metamorphosis occurring, and a minute portion of gangrenous lung is detached, carried to the left auricle and ventricle, whence it goes to the brain, or possibly to some other part of the body. But, wherever it goes, it carries the putrefactive element with it, which will be communicated to any part in which it may chance to be lodged.

As we have already alluded to hyperæmia and collateral œdema as the second cause of anæmia (*vide* post-mortem in hyperæmia, also causes of anæmia), very little remains to be added in this connection; but there is still one point necessary to be dwelt upon. In speaking of excessive hyperæmia and its sequel anæmia, mention has been made of collateral hyperæmia often following this or any other form of anæmia (*vide* prognosis of hyperæmia). It may be that the correct interpretation of this expression has not been seized, and hence an elucidation of what is meant becomes necessary. This collateral hyperæmia is nothing more than a diversion of the blood, the simple result of some other pathological condition. Supposing the quantity of blood to remain the same in the brain when in a strictly physiological condition, it is evident, on the other hand, that if by pressure during certain morbid processes the blood is forced out of one part, there will be an undue accumulation of it in another. This is collateral hyperæmia.

We have already seen how hyperæmia produces collateral œdema and resulting anæmia. It now will readily be perceived that anæmia may, in turn, produce collateral hyperæmia of other or adjacent parts; which again, according to its intensity, may terminate in collateral œdema; and often as a consequence of this œdema we have a 'secondary anæmia. Hence, whenever there is an anæmia in one part of the brain, the patient is endangered by collateral hyperæmia of another portion. Collateral hyperæmia might therefore be added to the causes of anæmia of the brain.

Anything producing undue pressure within the cranial space will cause anæmia of the brain. The particular manner in which

the capillary compression and subsequent anæmia are brought about will hereafter be considered. For the present it is sufficient to concede the possibility of a partial pressure on the brain. The question naturally arises, "How is it possible to have a particular or limited pressure? Pressure being continuous from molecule to molecule, it will not be localized." This conclusion is erroneous, having been arrived at by fallacious reasoning. It was supposed, as Niemeyer states, that the condition was analogous to that of a bottle filled with liquid in which a cork is being forcibly driven. The bottle will break at its weakest point, and not at the point where the pressure is directly applied. There is then a transmission of pressure, continuous and without intermission, and this pressure is applied to the whole bottle, and not localized. In this conclusion, as the last-mentioned author adds, a great fact was overlooked, viz., the anatomical division of the brain by the *falx cerebri*. The cerebrum is essentially a dual organ, and is thus equally divided into hemispheres; and by still another membrane, the *tentorium cerebelli*, the encephalon is subdivided into three portions.

Now, it is evident that if pressure of a certain degree (pathological or traumatic) be applied to one of these distinct portions of the brain, the membranes above mentioned will effect a limitation of it to the hemisphere to which it has been originally applied, the other hemisphere remaining completely unaffected. This same law applies to the cerebellum, which may be diseased or suffer from hemorrhage, without the cerebrum being perniciously influenced, though not invariably so; as it may happen that the pressure applied may be very great and thus extend beyond a certain limit, when, of course, the membranes would yield. This occurs very seldom. It is clear that sometimes a circumscribed pressure exists in the brain, in no degree transmitted to distant parts; and in this case you will understand that a tumor, for instance, can exercise pressure, and produce regional symptoms due to a partial anæmia.

In what manner does capillary compression (from abscesses, etc.) produce anæmia of the brain? In the simplest way possible. Imagine a sponge filled with water and resting in the palm of the hand. The moment the fingers are flexed upon it the sponge is compressed and the water exudes. In this manner will a clot,

abscess, etc., pressing upon the capillaries, force the blood from them, and produce anæmia in the immediate vicinity.

THE PATHOLOGICAL ANATOMY IN ANÆMIA OF THE BRAIN.

When speaking of the post-mortem appearances of the brain in hyperæmia, it was noticed that the result of our examination was often unsatisfactory and deceptive, and that, whilst looking for a hyperæmic brain, we might find one which was anæmic. These apparent anomalies and difficulties again meet us in anæmia. During life the anæmic condition of certain parts is readily recognized. For instance, in the face we note the skin, which, owing to its rich vascularity, is very prone to show us an anæmic condition, even while the vital processes are still active. But after death we no longer expect to see the healthy, rosy hue, but rather a pale, leaden, cadaverous look, and from this we draw absolutely no information in regard to excessive or diminished vascularity during life. In death, hyperæmic parts often look anæmic, and anæmic parts appear hyperæmic, from the gravitation of the blood and its settling after the cessation of circulation, and thus there are many causes which are apt to lead us into error. But we do know that, as a consequence of persistent anæmia, softening ensues, and that, if the anæmia be intense enough, even liquefaction may result. Therefore, softening is *the* condition we have to look for as corroborative evidence of anæmia, and it is easily determined by pouring a gentle stream of water on the brain, when the softened parts will appear more or less disintegrated, according to their different conditions, and be washed away.

It happens that the brain, in various forms of softening, presents either a whitish, yellowish, or reddish appearance; but there is nothing in this to claim our particular attention. There is another condition not to be overlooked, viz., hemorrhagic infarction. Here we have the brain dotted with a bloody tint, from minute capillary extravasation; but this is caused by embolism of one or more arterial trunks. In embolism there is always this tendency to capillary rupture and hemorrhage. This occurs in the lung or in other organs just as readily as in the brain; in fact, wherever the embolus happens to be arrested. This is only mentioned to warn the student from mistaking these minute extravasations for the appearances of hyperæmia when, in reality, anæmia exists.

The extent of the softened parts is exceedingly variable, being from the size of a pea to that of a hen's egg, or even larger. It sometimes happens that large portions of the brain are softened without any grave symptoms being manifested during life. This depends entirely upon the importance of the parts implicated; and although softening of the medullary fibres of the cerebrum might not be attended with many or any symptoms, it is evident that in other parts, as the medulla oblongata or cortical portions of the brain, softening would give rise to serious and unmistakable symptoms. In some cases of brain-disease, whether the patient dies from an acute or a chronic affection, we may often be sorely puzzled as to the diagnosis, and post-mortem examination may show nothing satisfactory to explain the symptoms which were evinced during life. In such cases it is always well to examine the middle cerebral artery, and we will frequently find it obstructed, more particularly so when sudden paralysis occurred during life. (See embolism and thrombosis.)

The anatomical condition in anæmia and collateral œdema is very important. The post-mortem examination will reveal, in some instances, a pearly, glistening lustre in the anæmic parts, and the scalpel will be wet or moist with serum.

Among the ordinary pathological manifestations of pressure is a marked depression of the sulci; the brain, being full of serum, tends to swell, and, as compared with a healthy brain, is remarkably white. Or we may have a protrusion of the *tentorium cerebelli*, or of the *falx cerebri*, which may dip to one side from pressure on the other, or the pressure existing in the lower part of one hemisphere may cause a dipping of the *tentorium cerebelli*. In both instances it will depend upon the amount of pressure, and in mild forms of disease may escape observation altogether.

SYMPTOMS OF SOFTENING OF THE BRAIN.

When speaking of hyperæmia of the brain, general hyperæmia has been considered. The causes of partial anæmia, followed by partial softening, are all that we have thus far analyzed. General anæmia will be investigated at a future time.

In considering the symptoms of softening, two important facts must be borne in mind: 1st. There are no pathognomonic symptoms of softening. 2d. The symptoms of anæmia and of hyper-

æmia are generally very similar. Consequently, a reliance upon any one symptomatic indication will, in most cases, lead you astray. Inasmuch as the treatment is of the highest importance, it is necessary to make a correct differential diagnosis and arrive at proper conclusions. This diagnosis can only be made by patient, deliberate, and careful investigation of the history of the case, and by becoming conversant with all the pathological and etiological facts and data obtainable. We have seen that, although this knowledge is very needful, the student must be careful not to be too exclusively guided or biased by any single deduction: whilst mindful of the pathological laws of softening, he must carefully weigh and consider the history and probable cause of the disease. With Niemeyer, I would urge you to have due respect for the sequence of events and symptoms in a given case. By the term sequence is meant the manner in which the events or symptoms succeed each other. This will often enable you to draw pretty correct inferences as to the probable cause of the disease. If the phenomena are characterized by suddenness, you may infer embolism; if, on the other hand, the symptoms have been slow in development, you may presume it to be thrombosis, provided youth does not exclude the conclusion. To obviate every source of error, I will successively take up all the forms and corresponding symptoms of anæmia.

The following law must first be established: whenever there is a sudden shutting off of a considerable supply of arterial blood from a part of the brain, there will be an interruption in the functions of that part which will be in direct proportion to the amount of blood excluded. If the blood-supply has only been diminished, a partial or limited suspension of the functions of the part will be the result; but if the supply be completely shut off from an important artery, there will be an entire suspension, in fact, an abolition, of the physiological actions of the part involved.

In anæmia of the brain we find two sets of symptoms: symptoms of *excitation* and of *depression*. These symptoms may exist separately, or in combination, or may follow each other in alternation. This last is the case where the blood-supply has not been entirely suspended. As the disease advances and the calibre of the artery grows smaller, the symptoms of excitation diminish, while those of depression increase. Where the symptoms of

depression alone exist, and persist, we may infer a complete shutting off of the arterial supply.

The symptoms of excitation are: increased or preternatural excitability of motor, sensory, or psychical functions, headache, hyperæsthesia of the auditory nerve and of the nerve of vision, with consequent photophobia, pain and symptoms of irritation in different parts of the brain, and an implication of all the nerves of special sense. The patient may feel unusually active and quick of perception, and be very bright and unusually cheerful. This may be followed by a feeling of lassitude and inactivity, the state of depression. In this state (of depression) there is generally—1st, an impairment of the mental faculties: one of the most easily recognizable symptoms of this is loss of memory. 2d. There is a stolid condition of the individual, a hebetude, accompanied by an appearance of lassitude and disinclination to exertion, eventuating in mental imbecility. 3d. The judgment becomes impaired, and all the mental faculties will gradually be noticed to have undergone a retrograde change, finally becoming entirely lost or annihilated. These different affections are generally progressive, and we will soon observe, more or less completely developed, the symptoms of amnesia (loss of memory), complicated with agraphia and aphasia.

In agraphia there is an inability to write. This condition does not depend upon any muscular impediment, but is simply the effect of amnesia; the patient has forgotten the alphabetical characters or the art of combining these into words. Aphasia is a partial or complete inability to speak or converse. Here there is no difficulty of articulation, but there is a want of ability to recollect the words which are required to express an idea. When certain cortical cells are entirely destroyed, it is impossible to reproduce the memory of ideas.

And so with the affections of the motor system. If the attack be sudden, motion is usually at once destroyed. If it be gradual, we have, first, a limited paralysis, affecting only a few muscles in the beginning; then the paralysis extends and attacks another part, and so on until gradually an entire and permanent hemiplegia will finally result. I say *hemiplegia* because, as yet, we are dealing with *partial* anæmia.

Now, what are the symptoms peculiar to anæmia the result of

thrombosis, embolism, collateral œdema, or pressure upon the capillaries?

THROMBOSIS.

Thrombosis, unless relieved, ends in softening of the brain, which is the result we are to anticipate. How are we to recognize this disease, either in the recent or the advanced stage? Though sometimes a difficult matter to make the diagnosis, it is in most cases a possibility; and although you have no absolute certainty, you will nearly always recognize the leading features of the affection. Of course you have to keep well in mind everything connected with the developments, and be very careful in analyzing the different facts.

Thrombosis, a result of a diseased condition of the arteries (*endo-arteritis deformans*), usually occurs in persons quite advanced in years. Hence the age is a significant fact, which must not be lost sight of in your examination of patients. You would hardly think of diagnosticing thrombosis in a young, healthy, vigorous man; but where the patient has passed the meridian of life, such a diagnosis would have just grounds to rest upon. But, as age alone is not a sufficient fact upon which to base your diagnosis, you must have recourse to other considerations.

Great importance has been attached by some authors to the condition of the peripheral arteries, as a diagnostic sign of thrombosis. Of course it is probable that if a diseased condition of the arteries exists in the brain it may also exist in other portions of the body. This may be the case *at times*, but it is by no means invariably so. We may have *endo-arteritis deformans* in the cranium and yet not have it in any other portion of the body, or, *vice versa*, it may exist in some other organ and still be absent in the brain. So that the evidence furnished by the diseased condition of the peripheral arteries may sometimes be *confirmatory*, but nothing more.

The condition of atheromatous degeneration of the arteries in any accessible part of the body is not very difficult to recognize. We generally have in affected vessels a peculiar atheromatous, retrograde, inflammatory condition,—the artery being preternaturally tortuous, rigid, and unyielding, without resiliency, and offering to the touch of the experienced observer an inelasticity the peculiarity of which he will immediately recognize; though if the

patient has exhibited *chronic* brain-symptoms in connection with a certain advanced age, and superadded to these we have the condition of the peripheral arteries just alluded to, there is then strong presumption that thrombosis exists.

Still, this presumption is not a certainty, and we need one link more in the chain of pathological evidence to render our diagnosis complete. This will be furnished by a study of the positive and rational symptoms, which are those of irritation and of depression,* and these symptoms will alternate or vary in obstinacy or intensity. Paralysis comes on, leaves, returns; a patient will be very bright one day, and correspondingly dull the following day. This variation has been *incorrectly* considered to be pathognomonic of *softening*. It may be characteristic of anæmia, but certainly not of softening; which latter condition, being a result of anæmia, is persistent, admitting of no possible variation of symptoms. In softening, the return of the physiological functions is utterly impossible: they are *entirely destroyed*.

These *variations* of symptoms, especially in paralysis, indicate *only a disturbance of the circulation in the brain*, and simply point to alternations in the extent of the anæmia. But when once a part of the brain is perfectly and permanently anæmic, it softens; and when there is *no* variation in the symptoms present, it points to total abolition of those functions which, for their proper performance, depend upon the integrity of the part of the brain presiding over their evolution. The disturbances of the circulation are due to the following facts: 1st, the gradual narrowing of the calibre of the arteries; 2d, the alternations of anæmia and collateral hyperæmia, the accession of which produces disordered vascular action, and corresponding symptoms. But in thrombosis the resulting anæmia is more slowly produced, as are also the changes in structure, causing a *gradual* development of symptoms, the manifestations of definite pathological lesions.

To recapitulate, we find in thrombosis:

1st. The advanced state of age. 2d. The gradation and duration of the symptoms. 3d. The variations of the symptoms. 4th. The morbid condition of the peripheral arteries, though not necessarily present.

* *Vide* page 45.

EMBOLISM.

Embolism is generally easily recognized, its symptoms being quite plain, and the facts apparent.

Suppose a man evinces symptoms of softening of the brain: embolism suggests itself. But without the characteristic *suddenness* of the appearance of the symptoms, you have no embolism. In order to obtain the confirmatory evidence, inquire at once into the history of the case; and should you ascertain that your patient has been subject to frequent rheumatic attacks, you will examine the heart, and, detecting a *bruit*, mitral or aortic (diastolic or systolic), will with surety diagnosticate embolism. Do not fail, therefore, to examine the heart and lungs, and should you find, in addition to the symptoms already enumerated, these organs in a condition favorable for the production of an embolus, you may with safety arrive at the above conclusion.

I have already told you that embolism occurs mainly with strikingly marked, characteristic symptoms, and that there is, therefore, less liability to be mistaken in these cases than in those of thrombosis; indeed, the suddenness of the attack almost alone enables us to diagnosticate with no little precision. There will be still less liability to err should you recollect the exact manner of production of embolism, which is, as you now know, very closely allied to the resulting symptoms.

In former lectures I have fully investigated the different causes producing embolism, having already stated that it is sometimes, though rarely, a result of thrombosis (caused by phlebitis or the puerperal condition), under which circumstances the embolus will be found in the pulmonary artery, and I need hardly add that instant death will certainly result should its entire calibre be obstructed.

It sometimes happens that embolism takes place in the spleen, or in the liver, either of which is very difficult to recognize during the life of the patient.

Embolism in the brain is of frequent occurrence, and here we have to deal principally with its results. I have also told you that the embolus is generally lodged in the middle cerebral artery, in the fissure of Sylvius, resulting almost inevitably in softening, owing to the non-establishment of the collateral circulation, being

situated above the circle of Willis. We have, therefore, no hope of recovery; for, should the patient not immediately perish, a hopeless hemiplegia will be the consequence.

There is a very important matter to which I wish once more to allude: it is the unfortunate use of the term apoplexy in connection with certain symptoms constituting the apoplectic state. Apoplexy, as commonly accepted, designates a particular disease (cerebral hemorrhage), and the synonymous use of the word is much to be regretted.

In a former lecture, when speaking to you of the apoplectic form of hyperæmia of the brain, I told you that the apoplectic condition is common to many different diseases. It consists in sudden abolition of consciousness, sensation, and voluntary motion.

This condition is always present in cerebral hemorrhage; but it also exists in cases of epileptic coma, anæmic coma, uræmic coma, etc., and in fact in many diseases which possess very different pathological starting-points. Therefore, when we say that a man is in an apoplectic condition, we do not necessarily imply that he has cerebral hemorrhage. As in the case of an ox felled by a powerful blow, there will be abolition of sensation, of consciousness, and of voluntary motion.

I will add that this apoplectic state not only *always exists* in embolism, but also constitutes a very marked feature of the disorder. A decided anæmia will result in the part which, by the obstruction of an important artery, is deprived of a large amount of blood. The consequence of this shutting-off of the vascular supply has already been considered,—the law being that the intensity of the disturbances will depend upon the more or less complete occlusion of the artery.

Hence, should the anæmia be complete, symptoms of depression alone will occur; if incomplete, we will observe an alternation of the symptoms of excitation with those of depression.

It is invariably of great importance to ascertain what disease has occasioned a suddenly-developed coma. Were the physician to confine his diagnosis to ascertaining the presence of *coma*, he would only expose his ignorance of the fact that this condition is but a symptom, and not a disease, and no more to be treated regardless of its pathological causation than would be a cough, a fever, or dyspnœa.

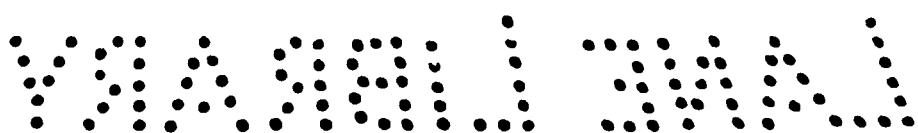
The apoplectic condition does not occur in thrombosis unless the latter be very extensive; and in the majority of instances thrombosis is so gradual that the apoplectic phenomena set in only towards the termination of life, closing the scene. The reverse will be observed in embolism: the same symptoms manifesting themselves *ab initio*, being, in truth, the first signs which attract attention and cause the physician to be summoned.

The first knowledge, then, of the presence of embolism proceeds from the apoplectic state which overpowers the patient. When these apoplectic phenomena cease (which, however, does not always happen), what will be the condition of the patient? You will find a well-marked hemiplegia of the right side of the body (the embolism being in the left middle cerebral artery); and the reason that the paralysis is situated on the side opposite the one affected is that the lesion is located above the point of decussation of the anterior pyramids of the spinal column.

This hemiplegia is almost always permanent. Indeed, I have never seen or read of an instance where the paralysis disappeared after an embolism: the reason of this being that, when once plugged, the artery is closed forever.

A cork driven into the neck of a bottle will form an illustration, with this difference in the conditions: that in the case of the bottle the cork may be removed; but the embolus cannot be extracted from the artery.

It may be that, many years hence, at a very advanced stage of science, there will be discovered certain means by which to dislodge or dissolve the pernicious obstruction: this, though most improbable, we will not deem impossible,—agreeing rather with Dr. Dunglison, who, after a long and rich accumulation of scientific experience, “had learned never to use the word impossible.” But, to return to the subject, the patient, should he live at all, will remain hemiplegic, and the apoplectic phenomena will pass off. You ask me why the consciousness returns, whilst the hemiplegia persists. Although I can give you no positive answer to this question, I believe, with Niemeyer, that the following explanation may be offered. The abolition of consciousness being due to the intense anæmia of the brain, collateral hyperæmia and resulting œdema follow, intensifying the apoplectic phenomena. After the lapse of a certain period of time, say a few days, this



collateral œdema is re-absorbed, and, consequently, the apoplectic state caused by the œdema rapidly disappears; after which re-absorption the pressure which was exerted upon the cortical cells of the convolutions of the brain is also relieved, these cells once more developing functions of ideation and of memory, and exhibiting intellectual activity.

Another important fact to be mentioned in this connection is that collateral œdema is a condition not necessarily confined to the brain. Hence, if you know how to apply its phenomena to this organ, you will not be at a loss to apply them in any other situation. In the spleen, or the liver, or other organs, embolism is followed by collateral œdema. Why this collateral œdema or why hemorrhagic infarctions follow embolism, we do not know.

You will now ask, How is it that, the brain being a dual organ, one part should be inactive, or even destroyed, and yet the other part remain with function? Why might not the right side compensate for the mischief created in the left? The answer is, that the collateral œdema is extensive, and its presence excessively deleterious, exerting more or less pressure upon the other hemisphere; and although, as I have told you, one of the functions of the falx cerebri is to prevent this encroachment, which it sometimes does successfully, in some instances it will be powerless in this respect, the falx then dipping over to the healthy side. The pressure, therefore, being transmitted, the dual action will be interfered with, and even temporarily abolished. It has been observed by Niemeyer that the posterior lobes are afforded better protection "against a pressure acting on the opposite hemisphere than the frontal lobes are, because the falx is much broader posteriorly, and hangs much farther down than it does anteriorly."

We have still another important fact to consider: it is that we often have coincident embolisms. Of course it must be only after strong presumptive proof that you diagnosticate embolism; but if you want confirmatory evidence, examine the peripheral arteries that are easy of access, and you may find one or several of them obstructed. This fact will sometimes prevent you from being led into error. Remember, therefore, that coincident embolism may or may not be present. Coincident embolism is caused when the clot of fibrin constituting the embolus, after becoming

detached, separates into several emboli, which are carried by the circulation into different arteries.

After having attentively listened to all this, you may still find it a very difficult matter to diagnosticate cerebral hemorrhage from cerebral embolism; and only by thoracic exploration can you arrive at a safe conclusion. Let us imagine two cases, both at the same time falling into the apoplectic state. The diagnosis will be based upon: 1st, a *history of the cases*; 2d, the examination of the chest; 3d, the sequence of events; 4th, the age of each individual. The age is very important, for we already know that cerebral hemorrhage generally results from atheromatous arterial changes, frequently themselves an accompaniment or a cause of thrombosis, which is a disease peculiar to old persons. Embolism, on the contrary, occurs at all ages, irrespective of sex or of other conditions of life. Yet in some instances obscurity exists, age not settling the question. In this disease, as in others, typical cases do not always solve the problem. It is stated by authors that even children are occasionally victims of cerebral hemorrhage. The rule, however, is that in cerebral hemorrhage age has great significance,—being the disease of persons past the meridian of life, the result of *endo-arteritis deformans*. We will now sum up the points of diagnostic difference between thrombosis and embolism:

THROMBOSIS	EMBOLISM
is always preceded by brain-symptoms;	is rarely preceded by brain-symptoms;
symptoms gradual;	symptoms sudden;
no rheumatic history;	oftentimes rheumatic history;
occurs in advanced age;	occurs at all ages;
<i>endo-arteritis deformans</i> of peripheral arteries sometimes occurs;	coincident embolisms are sometimes present;
apoplectic phenomena during the last stages.	apoplectic phenomena <i>ab initio</i> .

Never take any one symptom as a positive evidence of a disease. Do not confide too strongly in typical descriptions. I have never seen a case of typhoid fever which strictly corresponded in every particular with the typical delineation of the authorities. You must rely greatly upon the care you exercise in studying disease, and upon your earnest efforts to diagnosticate properly. Do

not essay more than you can accomplish, thereby becoming careless, and prescribing for various symptoms without arriving at their proper interpretation. Diagnosis first, therapeutics afterwards.

You cannot always leave the bedside with the perfect satisfaction that you have mastered the question of your patient's ailment: the most accomplished practitioners sometimes fail to ascertain in one visit the true cause of a malady, and, indeed, several may be made before certain diseases can be recognized, if recognized at all. Again, I am satisfied there are some affections which cannot be brought under any nosological arrangement. Hence you should endeavor to become good diagnosticians, and, when you are unable to classify a disease, should apply to its treatment the principles derived from your general pathological and therapeutical resources.

Should you visit a patient for the first time in consultation with another physician, do not let the latter imbue you with his ideas before making an examination for yourself, or you will almost certainly be biased.

SYMPTOMATOLOGY OF ANÆMIA FROM COLLATERAL ŒDEMA.

How may we recognize anæmia as the result of a collateral œdema in the brain? In order to do this, we must be guided by the following facts. Whenever we have a partial anæmia, the result of a collateral œdema, the symptoms of the one will be complicated by the symptoms of the other. We have seen that collateral œdema is sometimes due to embolism; yet, again, we know that hyperæmia of the brain will sometimes have the same effect. In collateral œdema following embolism, a capillary anæmia will be produced, which, if not relieved, terminates in softening. Furthermore, when a collateral œdema, the result of hyperæmia (which has not yet resulted in permanent partial anæmia), occurs, we will find the symptoms of collateral œdema accompanied by those of the *primary* disease.

All the pathological variations already alluded to, partial hyperæmia, collateral œdema, partial anæmia, softening, etc., are produced by tumors, clots, abscesses, or anything that will persistently encroach upon the substance of the brain or disturb the circulation of the blood therein. When the symptoms of hyperæmia present themselves, no matter by what cause produced, the secondary

phenomena will be readily understood, and the ultimate result may be softening of the brain. Now, when you have a patient presenting these symptoms, with a violent collateral œdema of the brain, will you attack that œdema with diuretics, or absorbents, or derivatives? Most assuredly not; but you will endeavor, by directing your attention to the *primary* affection, to relieve the hyperæmia, by which you may prevent softening. In other words, treat your patient anti-congestively, so to speak, and you will at least palliate, should you not cure, the disease. But if, by an unpardonable oversight, you were to treat certain symptoms, perhaps those of the secondary anæmia, you would of necessity make matters worse; for you would thereby intensify the hyperæmia, and a hopeless softening would surely result. You therefore readily understand, and of course appreciate, the necessity of dwelling upon so important a point. Hence remember that, should you diagnosticate a secondary anæmia, resulting from a previous brain-disease, or from anything else imaginable, the symptoms will be blended with those of the primary affection, whatever that may be, which it is your business to be able to recognize, and to treat accordingly.

This collateral œdema enables us to explain several obscure forms of disease; for instance, it must have occurred to those of you who have witnessed post-mortem examinations that the appearance of the brain is sometimes quite different from what we expected to find it, judging from the symptoms present during the life of the individual.

Let us suppose, for example, that you have a patient suffering from hemiplegia, a paralysis either of motion or of sensation, or of both, with or without spasms, etc. After death you examine the encephalon, and all you find may be a small clot lying upon the superior part of the periphery of the brain. Now, how will you proceed to explain that a small clot, situated upon the surface of one of the convolutions of the brain, will produce paralysis? You will answer, By the pressure the clot exercised upon the convolutions, which pressure is transmitted to the medullary masses, and finally to the thalamus opticus, the corpus striatum, or even to more distant parts at the base of the brain. Such, however, would be an incorrect conclusion; for, if you recall what I have already stated, you will remember that the brain is incompressible.

You have not, I am sure, forgotten the *explanation* of Niemeyer, that a clot so situated will produce an irritation, in the neighborhood of which you will have a hyperæmia, and, as a result of this, a collateral œdema, and finally anæmia; and this collateral œdema dipping deeply into the substance of the brain, and the *transuded serum* pressing upon the corpus striatum, a paralysis will be the result.

In this manner, then, are we enabled to explain how a clot upon the periphery of the brain may produce paralysis; and, in proportion as the peripheral clot becomes absorbed, the resulting hyperæmia will be removed, the collateral œdema disappear, and the symptoms of paralysis slowly and gradually pass away.

In addition to the foregoing, we have symptoms of other diseases, which can only be explained in this way: that is, by the pressure of a collateral œdema. For instance, the cerebellum may be almost destroyed without any peculiar symptoms necessarily revealing the fact during the life of the patient. Half a lobe may be destroyed, even an entire lobe be involved, or both lobes may be almost entirely gone, without much evidence of the fact during life. (It has been contended by eminent physiologists that the cerebellum is the seat of the power of co-ordination; but this I am by no means disposed to admit.) On the other hand, it happens that some of the most serious symptoms, such as hemiplegia, sometimes co-exist with the above lesions.

How are we to understand such paradoxical conditions? How shall we explain these seeming contradictions? The enigma is solved when we recollect that we may have a collateral hyperæmia extending upwards towards the brain, and followed by a collateral œdema. In the former case, a hemiplegia would be explained. Or the hyperæmia may extend towards the corpora quadrigemina by the superior peduncles of the cerebellum, the *processus e cerebello ad testes*; or it may be conducted along the middle peduncles, reaching the pons varolii or the tuber annulare; or, again, it may travel along the inferior peduncles to the restiform bodies, producing, in each case, corresponding phenomena, which may exist separately or conjointly, causing the most serious and complicated results.

We have but one more form of symptoms to consider in connection with anæmia of the brain: any force or foreign body, or

any imaginable pathological condition that will in any way encroach upon either hemisphere or the cerebellum, will produce disturbances of the circulation, and result in paralysis, hemiplegia, softening, etc. Should the pressure be on one side, we will have a hemiplegia of the opposite side of the body. Should a tumor or an abscess be present, the paralytic phenomena will probably be slowly produced, because there will be sufficient time for atrophy of the brain to prevent great encroachment on the surrounding mass on the part of these adventitious products. But the medullary masses of one hemisphere may be diseased and softened by tumors, abscesses, etc., and still no peculiar symptoms necessarily result. This is due to the comparative non-importance of these parts, and to their growth having been very slow and gradual, with a corresponding absorption of brain-substance as it became replaced by the intruding body. But in anæmia, which occurs when the tumor, etc., develops rapidly in proportion to the amount of destruction of the nerve-cells, you will find symptoms, speedily developed, of irritation and depression and brain-disturbances of a paralytic character. It is an important fact to remember, that these phenomena will depend greatly upon the situation of the parts involved. Were there no perplexing difficulties in the recognition of symptoms peculiar to diseases of the brain, their study might become more satisfactory. Experience teaches us that the diagnosis of all brain-maladies is quite obscure. There is one disease, however, the symptoms of which are, according to Niemeyer, unmistakable, almost pathognomonic, so that we are enabled to diagnosticate it quite accurately. I allude to an encroachment of some kind upon the posterior cranial fossa, that part which lies beneath the tentorium cerebelli. The symptoms attending diseases of the posterior cranial fossa are: 1st. Pains in the occipital region. 2d. Nausea. 3d. A morbid impairment of sensibility (sometimes anæsthesia, sometimes hyperæsthesia). 4th. A peculiar development of partial paralysis. 5th. Dysphagia. 6th. Dizziness possessing certain distinctive peculiarities. We all know that dizziness may be the result of an indigestion, or of an over-indulgence in fermented or distilled liquors, or may proceed from various other causes existing wherever vertigo is present, whether originating in the stomach, brain, or any other part. But in these last-mentioned cases the dizziness is always

subjective, being an hallucination,—patients imagining that surrounding objects are continually whirling around them,—and in any and every position, whether lying down, standing erect, or walking, the dizziness will continue without interruption; whilst, on the other hand, in diseases of the cerebellum this dizziness exists only when the patient is walking, or in the erect posture, and immediately disappears on his lying down. The dysphagia is ascribed to the implication of the glosso-pharyngeal nerve.

One more subject only have we now to consider in connection with partial anæmia of the brain, which is the treatment.

After all this monotonous talk about anæmia, you would undoubtedly like to be refreshed with a brilliant dissertation regarding the best modes of its treatment, with a careful comparison of their relative values. In this respect I must disappoint you, since the most I can say is, that *the less you actively interfere, the better!* A thrombus, you are aware, cannot be mechanically removed; nor have you any therapeutical resources at your command with which to attain the same end. Several things—stimulants, for instance—have been recommended, but they are all useless: for they can never unplug the artery. But there is one thing to which you must, nevertheless, not fail to resort: it is, the best hygienic treatment possible, using tonics to counteract loss of vigor. You may also treat symptoms that may arise, and, above all, must endeavor to obviate collateral hyperæmia.

Notwithstanding your patient may have anæmia, death from a collateral hyperæmia may occur. Should the symptoms of depression, which always exist in anæmia, be complicated by alarming symptoms of irritation, beware of collateral hyperæmia, and treat your patient accordingly, blistering, leeching, purging, etc.,—with due caution, however,—and you will at least succeed in averting the immediate and transitory danger, which would otherwise have resulted fatally.

LECTURE IV.

GENERAL CEREBRAL ANÆMIA.

Symptoms.—Diagnosis.—Treatment.

GENTLEMEN,—This condition most frequently originates in causes not restricted in character, but which are expressive of particular states of the system at large. All hemorrhages, excessive or colliquative discharges, whether menstrual, intestinal, hemorrhoidal, uterine, suppurative, or exudative, but especially the first mentioned, which are by far the most common, must sooner or later influence the production of an anæmia, more or less profound, from which the brain will suffer; entailing thereby a disordered and imperfect performance of its functions. The physiological law already alluded to must not, in this connection, be lost sight of. If the cause resulting in the abolition of the nervous excitability is not sufficient to effect its perfect destruction from the commencement, its abrogation will invariably be preceded by a condition in which will be observed an exaggeration of functional activity. This furnishes an explanation of many of the phenomena of cerebral anæmia, and offers the only rational interpretation of the alternation and combination of the symptoms of irritation with those of depression, common to this affection, and which will lead inexperienced physicians to suppose that they are dealing with the manifestations of congestion, which they treat actively and with disastrous results, while, in reality, anæmia, more or less profound, lies at the bottom of the whole difficulty. The *hydrocephaloid* of Marshall Hall is an occurrence which will exemplify what we desire to designate as a common source of error in this respect,—its etiology being frequently misunderstood, and the therapeutic resources directed to its relief oftentimes, from their mistaken conception, increasing the evil they are intended to avert.

Fevers have most pernicious effects on the constitution of the blood, and produce and intensify anæmia by their combustion and

rapid oxidation of the tissues, the results of which are familiarly evinced during convalescence from protracted disease, and by the "delirium of inanition," described by Dr. Clymer in "Aitken's Practice."

Quantitative and qualitative blood-changes will, more or less, influence materially the production of cerebral anæmia, especially when from chylopoetic disease, or the different dyscrasiæ, improper or deficient hæmatosis results. The absence of properly arterialized blood—its presence being a *sine qua non* for the proper performance of the cerebral functions—will be immediately appreciated.

It follows, from what we have said, that anæmia is produced whenever the brain receives an insufficient quantity of blood, whether the result be due to arterial obstruction or to reduction in the volume of this fluid. Moreover, the oxygen of the vital current, being an essential agent, must not be unduly diminished, otherwise the proper nutritive processes will not be accomplished, and the normal performance of their functional activities will be prevented. The red corpuscles being the "carriers of oxygen," a deficiency on their part, so far as the brain is concerned, would be equivalent to an insufficiency of the blood itself. Jaccoud and Niemeyer mention, among the causes of general cerebral anæmia, those of vascular origin, oftentimes reflex in character, produced by unusual mental emotions, eventuating in a contraction of the cerebral vessels. In these cases, loss of consciousness and sudden pallor of the countenance constitute the prominent symptoms.

Mechanical causes are also cited, as in instances where an undue amount of blood is retained in other organs at the expense of the general circulation. It is a well-known fact that syncope is frequently caused by the moving of patients ill with fever, or where they assume too soon the erect posture while convalescing from protracted and exhausting affections. Dr. Todd, in his work upon "Acute Diseases," particularly insists upon the serious dangers attendant upon exertion in both instances. The hazard is enhanced by a combination of such causes as an enfeebled cardiac impulsion and the sudden accumulation of blood in the lower extremities.

The symptoms will vary, according to the slow or rapid development of anæmia. In the latter instance, the phenomena of syncope—perhaps accompanied by convulsions, if the anæmia be

intense—may be anticipated. In cases of slowly-produced and more permanent cerebral anæmia, the symptoms of irritation will be more or less prominent. In graver conditions of this pathological state, marked symptoms of depression will succeed those of excitation, which are more temporary and initiatory in character. These latter will so closely resemble the phenomena presented by cerebral hyperæmia as to be inseparable in description; while the former will present more particularly such occurrences as syncope and physical and intellectual apathy.

The symptomatic manifestations of hyperæmia and anæmia, being identical, furnish no clue by which we can recognize and differentiate two pathological conditions diametrically opposite in character. The history of the case, concomitant symptoms, and general condition of the patient's system will therefore furnish the only reliable data upon which to base our conclusions, which would necessarily be erroneous were we to rely exclusively upon the cerebral symptoms, or even attach undue importance to the appearance of the patient; having before stated that pallor may co-exist with profound and dangerous hyperæmia, thus constituting a source of fallacy not to be forgotten. The clinical, pathological, and therapeutical antecedents must, therefore, be carefully studied. I can recall a case in reference to which for a few days I was in doubt, but finally diagnosticated cerebral anæmia, because the lady was greatly relieved when, in lowering her head, she favored the gravitation of the blood to her brain, and all her symptoms became remarkably exaggerated when in the erect posture. You who were with us last winter will recollect the successful issue of a case which I diagnosticated to be general cerebral anæmia, at the hospital, the result of a profuse hemorrhoidal flow, accompanied by epistaxis, which rapidly yielded to iron, digitalis, and a liberal administration of nutrients. Yet, owing to some striking symptoms of excitation, the case had previously been treated by several experienced physicians by spoliative measures, on the supposition of intense cerebral hyperæmia existing, with the result, I need hardly add, of greatly aggravating all the symptoms, and endangering the patient's life. Cardiac examination must never be neglected in doubtful cases, as an enfeeblement of the first sound or a diminished impulse, especially if accompanied by blood-murmurs, would have the utmost significance.

The mode of meeting the indications of a cerebral anæmia resulting from a sudden and profuse loss of blood resolves itself into the usual modes of relieving syncope. Position, arterial compression, even the temporary application of tourniquets upon the principal superficial arteries, Nélaton's method used in chloroform narcosis, consisting in holding for a long period the lower extremities high above the patient's head, the preparations of ammonia, ether and brandy, internally administered, and, in very critical cases, transfusion, are all to be judiciously essayed. In cases of habitual anæmia, chalybeate preparations, the improvement of the nutritive functions, an easily-digested and liberal dietary scale, especially of highly-nitrogenized substances, and the removal of the cause of the pathological condition, where possible, are resources of the greatest value, and will oftentimes be rewarded with success.

In conclusion, I would particularly warn you, gentlemen, to anticipate the dangers of *hydrocephaloid* disease in young children. Treat their diarrhœas—a fruitful source of this affection—early and earnestly. Do not be afraid of arresting the intestinal discharge for fear of producing brain-symptoms, as is too often done, in compliance with a maternal prejudice, fraught with danger to the little ones. Recollect, as Trousseau says, that the “continuance of diarrhœa in teething children predisposes to convulsions.” Do not be deluded by the supervention of the symptoms of irritation in children who have experienced colliquative discharges, notwithstanding the flushed face, heated skin, general restlessness, twitching, insomnia, and even convulsions. In such subjects, resort to stimulants and milk, and, in older children, to the famous “raw meat diet.” You will thereby prevent the subsequent stage of collapse, when the vital powers will be too prostrated to admit of recuperation.

Forewarned, you should be forearmed; and it will henceforth be inexcusable in you to commit such a blunder as to treat similar cases with spoliative measures. Resist all temptations to be misled by the threatening aspect of the initiatory symptoms, so deceptive as to compel you to select therapeutic means which would inevitably result fatally, consigning to a premature grave the little sufferer committed to your care; which catastrophe, instead of averting, as you should do, would be precipitated by an ignorance as unpardonable as unjustifiable.

LECTURE V.

MENINGITIS.

Acute Idiopathic Meningitis.—Pachy-Meningitis.—Tubercular Meningitis.—Cerebro-spinal Meningitis.—Simple Idiopathic Meningitis.—Characters.—Symptoms: Chill, Fever, Headache, Delirium, Vomiting, Constipation.—First Stage.—Second Stage.—Pericarditis.—Pneumonia.—Rheumatism.—Typhus and Typhoid Fevers.—Syphilis.—Hydrocephaloid.—Prognosis.—Causes.—Convulsions in Children.—Treatment: Drastic Purgatives, Cold Applications, Ergot, Bromide and Iodide of Potassium, Vesicants, Venesection, Leeches, Cupping, Counter-irritants.

GENTLEMEN,—We will now consider a class of affections of which delirium is always a prominent symptom, adopting Da Costa's method of grouping the nervous diseases according to some particular common symptom. Among the great variety of acute affections of the brain, which delirium always helps to characterize (being almost one of their pathognomonic symptoms), we have—

1st. Acute Idiopathic Meningitis. 2d. Tubercular Meningitis. 3d. Cerebro-spinal Meningitis. 4th. Delirium Tremens.

In the consideration of these diseases it is not necessary that we should fully develop all their accompanying details, having reviewed with great precision many common to all when speaking to you of hyperæmia and anæmia.

We, therefore, have only to apply the laws established, making but slight reference to facts already fully explained.

The disease which I will place before you to-night is that form of meningitis called

ACUTE IDIOPATHIC MENINGITIS.

The term meningitis signifies an inflammation of the membranes enveloping the brain. These membranes are called meninges; and, by affixing *itis*, which denotes inflammation, the word meningitis is formed (as in bronchitis, pleuritis, etc.).

Meningitis presents a few peculiarities, as the following:

The inflammation may be limited to the convexity of the membranes, as in acute idiopathic meningitis; or it may attack the base, as in tubercular meningitis; or the dura mater may be the only membrane implicated, as in pachy-meningitis; or the pia mater and the arachnoid may be the involved membranes.

These distinctions should be remembered, and, in order to impress them upon your memory, I shall recapitulate.

Pachy-meningitis is an inflammation of the dura mater only, and seldom, if ever, idiopathic (that is, produced without any apparent cause): being generally the result of an injury.

Idiopathic meningitis is an inflammation of both the pia mater and the arachnoid, of which I shall soon treat.

Tubercular meningitis is a most fatal malady, and usually a disease of children; attacking children ordinarily above two years of age, and principally those of a strumous diathesis. It may almost be called an incurable disease; for, notwithstanding a few cases of its happy termination are on record, an incorrect diagnosis is not at all improbable.

Cerebro-spinal meningitis, according to some authors, should not strictly be classed among the nervous diseases, since it is claimed to be a result of blood-poisoning; and that it is only a form of essential fever, the inflammatory products of which are in a measure directed to the nervous system.

Simple (acute idiopathic) meningitis is a disease which may occur at any period of life, attacking adults as well as children, and is always a grave and dangerous affection. It is sometimes with difficulty differentiated from other nervous complications. In children over two years of age the meningitis is apt to be of the tubercular variety; a fact of great clinical importance, for the reason that the patient sometimes recovers from simple, but never from tubercular, meningitis. Hence the diagnosis of the latter will be the death-knell of your patient, bringing dismay to the agonized mother as she tremblingly awaits your gloomy verdict.

It is my desire to enable you to recognize fully this disease without embarrassing your memory with lengthy and burdensome details. What you must know are the ordinary and prominent symptoms.

The initiatory phenomenon is a chill in the adult, and a convulsion in the child, as is usually the case in all acute inflamma-

tory affections. After this, reaction will take place, and the febrile exacerbation will be more or less intense. The temperature does not run so high, however, as in many other fevers, seldom rising above 102° or 103° F.; neither does it present any marked intermissions, but is continuous, differing in this regard from tubercular meningitis, which, like malaria, is accompanied by fever of a remittent type. It occasionally happens that these two last diseases are confounded.

After chill and fever, the next symptom found in simple meningitis is *headache*,—not of an ordinary character, but a violent, distressing, unmistakable headache, resulting in persistent insomnia, and torturing the patient by day and by night. His screams and entreaties are painful beyond expression to a sympathetic person.

The symptoms do not necessarily follow the order of sequence which I have given; you need not, therefore, be embarrassed in your diagnosis on hearing that, instead of the chill preceding the fever, headache, etc., the symptoms have all manifested themselves at about the same time.

A characteristic *delirium* follows the headache, perhaps without exception, even in elderly persons, who have experienced little or no fever. Its peculiarity is, that it does not partake of the loquacious, good-natured character of delirium tremens, but is so violent and furious that the patient is with difficulty kept in his bed, and in his rage he often endeavors to butt his brains out. It has actually happened that some have succeeded in their attempt at self-destruction, when carelessly watched.

Another characteristic symptom in meningitis is *vomiting*, the peculiarities of which may sometimes give you the clue to the diagnosis.

A great difference will be observed in the vomiting which accompanies gastric, hepatic, or abdominal disorders, and the vomiting in meningitis. How, for instance, does vomiting take place in bilious attacks, in gastritis, hepatitis, etc.? There is usually nausea, and the vomiting itself is painful and accompanied with straining; the patient is greatly distressed, throwing up all his food by violent spasmodic efforts. By the mere pressure of the finger upon the epigastrium, the pain will be increased and vomiting aggravated.

The phenomena in brain-diseases offer very distinctive features; the vomiting occurring without the slightest effort on the part of the patient, almost spontaneously in fact, and unaccompanied by pain or nausea,—features stamping it as characteristic of, and sympathetic with, disease of the brain. The vomiting resulting from gastric affections is generally relieved by the application of a mustard-plaster over the epigastrium. In cerebral trouble, however, the plaster should be placed upon the nape of the neck.

I must not neglect to mention another symptom, which is *constipation*, the bowels ordinarily being quite difficult to move.

We have now considered the principal symptoms of importance characterizing the first stage of the disease. You should remember them, since they are not always described in this order. You will find these symptoms far from corresponding accurately with the typical descriptions. In fact, you need not expect the first, second, and third stages to succeed one another in regular turn.

It is only an arbitrary custom thus to divide some maladies, which custom I have followed in order to make matters clearer to your minds, if possible. Consequently, be prepared to find upon your first visit perhaps the characteristics of every stage of the disease existing at the same time.

In affections of the brain, symptoms of irritation usually appear first, and are succeeded by those of depression, though this order is not absolute, the symptoms of depression being sometimes initiatory, as observable, for instance, in a patient stricken down by an attack of cerebral hemorrhage,—where you will have the comatose state from the commencement. In proportion to the intensity of the attack will you find the tardy or rapid succession of the symptoms. When extremely violent, as in the *méningite foudroyante*, those of depression set in at once: you find your patient comatose on the first visit, and dead on the second. But in milder cases the different stages are better marked, symptoms of irritation often existing for several days prior to those of depression or coma. And this latter condition is always to be apprehended; for it is usually of fatal termination. In these cases, the delirium resolves into stupor; you shake your patient to no avail: you cannot arouse him; the stupor deepens into coma,—his last sleep.

The corresponding symptoms keep pace with these changes;

and *pari passu* the pulse becomes frequent, feeble, irregular, and jerking, the skin is dry and parched, the breathing stertorous, and the patient's vital powers rapidly give way. He dies of the same coma which we find in cerebral hemorrhage, in epilepsy, or in uræmia, etc., except that in epilepsy it is transitory. This coma is the second stage of meningitis, and is an adynamic coma. I use the word *adynamic* here as we use the term typhoid to express the low ebb of the vital powers, the glazed tongue and feeble pulse, as found in typhoid fever, but applied also to that condition present in other diseases.

Watson graphically describes the manner of dying by apnoea, asthenia, anæmia, and coma. In meningitis, the patient dies of coma; and you will agree that it is much more simple to state this fact than to lecture two evenings upon the details connected with the second stage of that disease. If you remember all the points already given, you will have little difficulty in forming a clear idea of meningitis.

You will perhaps ask, How, with this grouping of symptoms, will it be possible for us to differentiate between meningitis and other diseases of the brain? It is possible for any man to make mistakes in this as in other diseases; but the common blunders which you would be liable to make if not forewarned I will endeavor to guard you against.

The most important point, in my estimation, is the diagnosis. A disease which you might take to be simple meningitis, and which has misled some good diagnosticians, is—

PERICARDITIS.

In this disease there are indications similar to those of the foregoing affections, viz., the head-symptoms. Should you make a hurried examination, you will at once conclude that you have a case of meningitis, and will immediately have recourse to active treatment,—purging, bleeding, etc.,—a treatment not quite in accordance with the modern mode of management of pericardial inflammation. I advise you to guard against this fallacy, and in such cases always to examine the thorax.

I once had a patient in whom several nervous symptoms presented themselves,—headache, vomiting, etc.,—and I was almost inclined to believe it to be a case of meningitis; for although the patient

had pains in the præcordial region, he referred the severest pain to the head. Certain disturbances of the pulse and of the respiration, however, soon put me on the proper track to diagnosticate pericarditis.

Pneumonia is another malady which sometimes presents symptoms similar to those of meningitis, the auscultatory evidence in the first stages of the former disease being frequently unsatisfactory. I have seen a patient who presented several suspicious symptoms of pneumonia, but I was unable to detect a crepitant râle, and there was no dullness on percussion. I finally examined the axillary space, when a distinct crepitation was manifest.

When you are puzzled as regards the disease, to know whether it be meningitis, pericarditis, or pneumonia, always recollect that delirium usually accompanies a consolidation in the apex of the lungs, and that auscultation will furnish the desired information. Experience has taught me that the least medication used in pneumonia the better, since it is a strictly cyclical ailment; whilst in meningitis active treatment, on the contrary, may be of benefit.

Another source of error arises in some cases of *rheumatism*, where the blood-poison is expending its virulence upon the brain, or perhaps a kind of metastasis supervenes. I well remember having suddenly lost a patient once with rheumatism accompanied with brain-symptoms like those of meningitis.

Several examinations of the brain, in parallel cases, have been made by Trousseau, without discovering any confirmatory evidence of cerebral inflammation, where death was preceded by well-marked meningeal symptoms. The efforts of other investigators, however, have been attended with contrary results. In rheumatism with an existing tendency towards the development of such complications, it is best to be ever upon your guard; never being over-confident in your prognosis.

Another set of head-symptoms intimately connected with the progress of *typhus* and *typhoid fevers* may also be mistaken for those of meningitis.

Typhus or "ship" fever is a disease only exceptionally seen as far west as St. Louis. In typhoid fever, especially in very nervous persons, the head-symptoms are quite prominent, and there is considerable nervous irritation. The Germans call this disease "*Nervenfieber*." To treat this fever actively would be a most

unfortunate and unpardonable blunder: therefore examine the patient's abdomen, and ascertain whether or not it is tympanitic and painful, or if pressure gives rise to gurgling and localized tenderness in the right iliac fossa.

Syphilis also is said sometimes to produce marked symptoms of cerebral irritation; and still another disease is hydrocephaloid. Of this I had intended to speak more at length in this connection, but shall defer it till reviewing the tubercular form of meningitis.

The *prognosis* in idiopathic meningitis is very unfavorable; so be cautious in giving your opinion, for the contingencies are such as to invest the result with great uncertainty.

The *causes* are *excessive mental fatigue; over-exposure to the rays of the sun; severe blows on the cranium; rheumatism*, and, perhaps, *syphilis*. As regards the morbid appearances of this disease, you can find them described at length in the books; but recollect that the dura mater is not involved, and that the condition of the pia mater and of the arachnoid depends upon the intensity of the attack. There is an exudation of pus, and more or less thickening of the membranes, which present a pearly and opaque appearance. The purulence occurs sometimes very early. I have read of cases in children where it was discerned within twenty-four hours from the commencement of the disease.

An important fact is, that in very young children the disease is not ushered in by a chill, but by convulsions,—a result partially due to their liability to convulsions; which fact, West says, is in consequence of the extreme mobility of their nervous system, which is therefore easily irritated by trifling disturbances, whether originating from teething, worms, fevers, or the exanthemata.

As to the treatment, I have not much to say. I do not believe in the efficacy of tartar emetic, calomel, or repeated and copious venesections in the treatment of inflammation, having known iritis to occur during mercurial ptyalism. You can accomplish much by administering drastic purgatives; and I have great faith also in the good effects produced by cathartics, as derivatives, in certain affections of the brain. As a matter of course, they must be used with caution, or the result will be unfavorable; but there is a medium in all things, even in the administration of beef-tea or water. At the very beginning of the disease, a purgative

is certainly beneficial: the old "ten and ten" (calomel, 10 grs., jalap, 10 grs.) being probably as good as anything else you can use.

Cold applications to the head, by means of ice-bags, must not be neglected; but never fail to give precautionary directions about applying this ice at intervals, and moving it about; for I have seen cases where the scalp had been actually frozen. Ergot is of advantage in meningitis, by its action upon the vaso-motor nerves, causing contraction of the blood-vessels; and its action may be intensified by the addition of bromide of potassium. Iodide of potassium also bears a good reputation, being with many writers the remedy *par excellence*; it is to be given freely and boldly, especially where a rheumatic or syphilitic taint exists.

Should the disease become subacute, shave the scalp, and pustulate it with croton oil: this measure, in children especially, frequently has an admirable effect. In cases with sthenic symptoms, you might resort to venesection, or to the application of leeches to the anus, the Schneiderian membrane, or the occipital protuberance. Cupping, also, is very good. Then use counter-irritants; apply mustard plasters to the feet, wrap them in warm fomentations, and cover with blankets. In this, as in other inflammatory fevers, give judicious nourishment, like beef-tea, or other food easy of digestion and absorption; for in most of these troubles there is a moment of crisis which is liable to carry off the patient unless the vital powers have been sufficiently supported. If, in spite of all this, your patient should show signs of approaching death, you might even then venture to administer stimulants.

LECTURE VI.

TUBERCULAR MENINGITIS.

Acute Idiopathic Meningitis.—Anatomical Lesions a Peculiarity.—Acute Hydrocephalus.—Symptoms: Period of Invasion, Gradual Impairment of Health, Change of Habits and Temper, Headache.—Importance of Cephalalgia.—Stages: Slow Pulse, Suspicious Respiration, Cerebral Maculæ, Boat-shaped Abdomen, Flush and Pallor, Cephalic Cry, Remission in Fever, Increased Somnolence, Coma, Changes in Paralytic Phenomena.—Sources of Error: Billious Intermittent Fever, Typhoid Fever, Hydrocephaloid of Marshall Hall, Partial Anæmia.—Prognosis.—Treatment.

GENTLEMEN,—In my last lecture, when speaking of *acute idiopathic meningitis*, I remarked that one of its most salient and characteristic points is its occurrence in children generally under the age of two years or above the age of ten, or in adults. I also stated that in children acute meningitis commences with convulsions, and that their early appearance or recurrence is quite characteristic of this disease. Simple meningitis we found was an inflammatory affection, accompanied by fever, and hence readily discriminated from active hyperæmia of the brain, by placing the thermometer in the axilla. Convulsions preceding acute disease in children are simply premonitory symptomatic indications of greater or less trouble, but are significant of no particular pathological state. They are produced by the presence of worms, dentition, the exanthemata, epilepsy, or brain-affections; in short, they occur in all cases of acute affections, or where some reflex irritation has produced central nervous disturbances. If you have carefully retained everything I told you when describing simple meningitis, you will know that I also referred *en passant* to tubercular meningitis, stating that it usually attacks children *between the ages of two and ten years*. It, therefore, is a disease peculiar to childhood, and is generally treated of in works upon the diseases of children. It may occasionally attack adults of a scrofulous diathesis, or in whom tuberculous deposits exist in the lungs, mesentery, or other organs.

The peculiarity of this disease is, that the anatomical lesions differ from those of simple meningitis. The membranes involved in both diseases are the same, it is true, but the latter affects the convexity, while tubercular meningitis attacks the base, of the brain, whence the French derive their name of "*méningite de la base*." You occasionally find described a disease called *acute hydrocephalus*. In what respects does it essentially differ from simple acute idiopathic, or from tubercular, meningitis? It is only another name for tubercular meningitis. It is called acute hydrocephalus because the disease was so termed by Dr. Whytt, of Edinburgh, who originally described it as "acute dropsy of the brain." This name does not, however, express the original character of the malady itself, but only one of its results. Moreover, in all forms of tubercular meningitis there is an exudation of fluid into the ventricles, causing softening by pressure, and a kind of saponified, friable, œdematous condition of the surrounding brain-structure, while in simple meningitis these cavities are, as a rule, empty. The name, acute hydrocephalus, therefore, expresses only a result; and you might with as much propriety call typhoid fever the disease of Peyer's glands, though the ulceration of these follicles is but a consequence of the action of the peculiar *materies morbi*. Now, one of the principal lesions in tubercular meningitis is this dropsical effusion in the ventricles of the brain; but we have others just as important; as, for instance, the tuberculous deposit at the base of the brain, or in the meshes of the pia mater, or in the fissure of Sylvius. Acute hydrocephalus is, therefore, an improper appellation.

Perhaps it would not be unsuitable in this place for me to say that, in my humble opinion, it is inadvisable to designate a disease by the name of an individual. "Duchenne's disease" implies nothing in relation to its pathology or symptomatology; but progressive locomotor ataxia immediately conveys to the mind the principal characteristic features of the disorder. The same may be said in regard to "Bright's disease" for certain affections of the kidneys, and "Addison's disease" for certain lesions of the suprarenal capsules. Acute hydrocephalus is the older, and tubercular meningitis the more recent, designation for the same trouble.

What are the principal symptoms of tubercular meningitis? I say *principal*, because I have not time to consider the minutæ.

With Trousseau, I believe that too great importance cannot be attached to the *period of invasion* as a diagnostic point, which to understand is an important step in advance, being the first successful attempt at untying the Gordian knot of the question. In acute idiopathic meningitis, you remember, there is a stormy outset, the fever and brain-symptoms rendering the period of invasion strikingly alarming. But in tubercular meningitis the period of invasion is so very peculiar that it cannot be mistaken by any careful observer. The child, being of strumous diathesis, of scrofulous parentage or ancestry, ceases to be healthy. It is not at once seized with tubercular meningitis, while enjoying perfect health, as in acute meningitis, but there is a *gradual failing of the general health*, and emaciation commences. This change, the beginning of which was scarcely noticed, at last becomes not only evident but striking. It is sometimes rapid, and the child, who perhaps was formerly fat and vigorous, the very picture of health, soon loses flesh, and becomes greatly emaciated. This change progressing, another symptom develops itself,—one on which Trousseau lays great stress. This is the moral change, manifested in the *habits and temper* of the child. The little patient, who was formerly perhaps of a playful disposition, grows irritable and peevish; objects which formerly were incitements to playfulness now cease to attract attention. The child becomes morose and disagreeable, indifferent to pettings and caressings, which formerly were eagerly sought for and fully appreciated, and experiences no interest or pleasure in the company of its little playfellows or in its accustomed frolics. At length the mother becomes seriously alarmed, and is positive in asserting that there is something the matter with the child; who very likely at this period begins to evince the existence of *violent headache*. This cephalalgia will come on paroxysmally, and the child, whilst running about, will suddenly stop, lean against something, put its hand to its head, and complain of violent pain. It will also be seized with *vomiting*,—not the ordinary vomiting from gastric disturbances, but true *cerebral vomiting*, which I described in my last lecture.

Of course the parents are now extremely solicitous, and a physician is sent for. Well may they be frightened; for although headache is not infrequent in children, still, when it follows slow emaciation and the other symptoms described, the mother feels that

there is probably some grave affection undermining the health of the little one, and her suspicions are well founded. If the physician summoned be the regular family attendant, he has undoubtedly already observed the nature and suspected the cause of the ominous changes in the child, and his case is pretty well made out before he arrives at the house. But if a stranger, and entirely unacquainted with the family, he will learn, upon inquiring into the history of the case, that the child is suffering from severe headache, perhaps vomiting, and, moreover, has been ailing for an indefinite period, and its health gradually declining. Do not lose sight of these facts: they are exceedingly important, and you must retain them, as they will prove serviceable. In order to prove the importance of cephalalgia in children as a symptom, I will relate to you a case which occurred in my own experience.

I was once called upon to visit a child suffering from severe headache. The attending physician was absent, and my immediate presence was desired. On my way to the house, it occurred to me that there might be something grave about this case, requiring, as it did, the immediate attendance of a medical man to relieve the suffering, not even awaiting the return of the regular doctor. On examination, I found that the headache had lasted several days, that contraction of the pupils existed, with photophobia, intolerance of sound, and marked fever, the temperature being 103° F. I immediately jumped at a diagnosis, which I caution you never to do; for the disease might have been remittent fever, which in children often produces violent head-symptoms, which are cured by quinine. But I felt justified in the conclusion I had so rapidly arrived at, because, upon inquiring concerning the previous health of the patient, I learned that it had failed gradually, that the attack had been insidious, and, as the mother expressed it, "she didn't exactly know when the child first got sick." After hearing this, reviewing the other symptoms, and revolving them in my mind, I was convinced; and, to my sorrow, I diagnosticated tubercular meningitis, without communicating my fears to the family. That same afternoon I met the attending physician, informed him of my visit to the child, and communicated to him my fears and diagnosis. He asserted that I was mistaken,—that the child was only suffering from a neuralgic affection. I reminded him that Dr. West states that neuralgia is of exceedingly rare occurrence

in children; nor would it be accompanied by fever, or preceded by slow and gradual deterioration of health. We parted, and a few days afterwards I was informed that my diagnosis had been correct, and that death had rapidly supervened. When you consider that Trousseau, with all his opportunities, immense practice, and enormous consultation experience, emphatically states that he met with only *two* cases of tubercular meningitis that recovered, and that even then it was highly probable that he had been mistaken in his diagnosis, you will readily be convinced of the hopelessness of the affection. Therefore, *beware of neuralgia* in children; for, generally, there is something more serious underlying the superficial symptoms.

Let us now consider the stages of this illness. I have already stated that if there be anything I abhor, it is the arbitrary division of disease into stages; as they so seldom correspond to the real accession and development of the morbid phenomena, which vary in different cases, untrammelled by absolute limitations. It is a source of fallacy, therefore; but, as it is the custom to divide disease into stages, I shall give you the different symptoms which, as nearly as possible, constitute those of tubercular meningitis. We have first the period of invasion, already described, with irritability, fretfulness, etc.; then fever, accompanied by a slightly accelerated pulse, headache, cerebral vomiting, wakefulness, constipation, and contraction of the pupils. These constitute what is termed the *first stage* of the disease.

We have here the symptoms of excitation, of hyperæmia, with the addition of fever. When this stage has passed away (always supposing that we have this regular succession of phenomena), the *pulse* becomes remarkably *slow*, falling to seventy, sixty, or even fifty per minute in very young children, and there exists likewise an interference with its rhythm. A peculiar *embarrassment of respiration*, not enough to amount to a dyspnœa or an orthopnœa, is produced; but there is a rhythmical disturbance which sometimes, though not always, corresponds to the change in the pulse. About this time a kind of mottling appears upon the skin, which has been described as *cerebral maculæ*, and upon drawing your finger over it quite distinct traces are left, which fade gradually. This is not, however, characteristic of the disease.

You will observe a *peculiar appearance of the abdomen*, consti-

tuting a sort of boat-shaped excavation; the intestines sink, the abdominal parietes shrink, and the superior spinous processes of the ilia come out in bold relief. This symptom, though not pathognomonic, is still quite characteristic of the affection, and should always be sought.

There are remarkable alternations of *flushing and pallor* present in all the varieties of meningitis, due perhaps to a peculiar state of the vaso-motor nerves, causing the alternate contraction and relaxation of the blood-vessels which they control; and when you examine the child, you will sometimes observe its face glowing and congested, and then there will be a sudden accession of marked wanness strongly contrasting. From these symptoms you will justly conclude that you are dealing with a case of meningitis, and that the danger is imminent and threatening.

There is also developed a *cephalic cry*,—a peculiar, piercing, wild shriek, never to be forgotten when once heard. After you have seen a few cases of tubercular meningitis, these different symptoms will become indelibly impressed upon your memory. The headache, flushing, pallor, sunken abdomen, stupor, disturbance of pulse and respiration, are quite characteristic, but, in reality, are merely the prodroma of the third stage,—coma.

In the second stage there is very often a *remission in the fever*, sometimes so marked that it may lead you to think you have made a mistake in diagnosis, and you will administer quinine, hoping to check the fever. But remember that this is merely clutching at a straw, as these remissions are characteristic of this disease, and may be observed even in the last stage, after coma has set in, and cause the mother to renew her hopes, which prove but too fallacious.

During the second stage a marked symptom is *increased somnolence*. In the first stage, owing to its great irritability, the child is annoyed by the doctor's presence, lights, sounds, etc.; but later it is sleepy, stupid, lethargic; the hyperæsthesia having left, you can pinch the child without its responding. After observing the delirium, maculæ, cephalic cry, headache, boat-shaped abdomen, flushings, peculiarities of pulse and respiration, and the remissions in the fever, you may imagine that there is no possible source of error. Bear in mind, as I have already told you, that it is not always possible to make a diagnosis at the first visit. You need

not commit yourself by giving a positive opinion, but should try, by proper observation, to arrive at correct conclusions, even if it take you several days. Also, keep well in mind the fact that these signs are common to different diseases, and do not attach too much importance to any isolated symptom. Judge only by the combination of manifestations, sequence of events, and concurrence of conditions, or you will be led into error.

The third stage is *coma*, as in simple meningitis. The pulse is exceedingly feeble and frequent, too frequent to be counted. There is perspiration and a clammy feeling of the skin, the pupils are dilated, and we have the symptoms of depression, with their ordinary paralytic phenomena.

These paralytic phenomena have this peculiarity, that they are inclined to change, so that we may first have a hemiplegia; then a paraplegia, strabismus, and ptosis may occur; or first one group of muscles will be paralyzed, to be followed by another, and then again the paralysis may entirely pass away. Occasionally there are convulsions, at other times they are absent; but if present, it is always towards the termination of the disease, while in acute idiopathic meningitis they always occur at the outset. The coma finally becomes more and more hopeless and intense, the pulse more frequent and feeble, the skin more clammy and moist, the urine and fæces are involuntarily evacuated, and death ends the scene.

Now, what are the diseases with which tubercular meningitis may be confounded? We have first *bilious remittent fever*. This we can readily discern by giving quinine,—a specific for this complication. If you are doubtful in your diagnosis, do not hesitate to administer this drug freely and boldly; for in cerebral affections it is best to give it in large and *sedative* doses, in preference to smaller ones, which only irritate and produce no compensating beneficial effects. Sometimes it becomes difficult to differentiate tubercular meningitis from *typhoid fever*, because the symptoms of the latter, especially those of a cerebral character, often bear a great similarity to those of the former. In children, the enteric symptoms—diarrhoea and tympanitis—are often absent, while the brain-symptoms are very prominent. But here you must remember the pathognomonic symptom,—the key to the difficulty,—which is the peculiar disturbance in the respiration, always present in

tubercular meningitis, never in typhoid fever. It is important to diagnosticate correctly in this respect, as typhoid fever is not often fatal in children, while tubercular meningitis is always an "implacable affection."

There is another condition to be considered in this connection, the *hydrocephaloid* of Marshall Hall. This is but another name applied to general anæmia of the brain, causing cerebral symptoms, which is developed in children after protracted exhaustive diseases, especially bowel complaints, vomiting, and diarrhœa lasting for a long time. It is very often the result of cholera infantum. In this disease, when the diarrhœa exists, the first thing in order is to check its progress. We should give brandy, and even ammonia, if the system will bear it; but we would inevitably produce disastrous and fatal consequences if we were to mistake it for congestion or inflammation, and treat accordingly these hydrocephaloid symptoms, which bear a striking analogy to the phenomena presented during the course of the meningeal affections we have just studied. On the contrary, gentlemen, it is under such circumstances that you must energetically sustain the vital powers. In this manner a cure may be effected; while, if these measures be neglected, death will surely follow. This proves the validity of an assertion made in a former lecture, of the similarity of the symptoms of cerebral anæmia and hyperæmia. In all these forms of meningitis there is an anæmia of the capillary vessels, produced towards the termination of the malady. The pathological law which I have so often given you can be once more applied in these affections,—I mean the state of depression which follows the primary one of irritation. In complete anæmia the abolition of the functions is absolute, and only the symptoms of depression are evident. But in partial anæmia we have the symptoms of excitation preceding those of depression; and this is exactly what happens in meningitis.

In conclusion, allow me to state that the prognosis is summed up in that small but comprehensive word—death.

As to treatment, there is none which has ever been successful. Notwithstanding this, you should spare no efforts to save the life of your patient; and I therefore recommend to you, as indorsed by the highest authorities, the iodide of potassium, freely administered in combination with the bromide of potassium. Use

counter-irritants, and pustulate the scalp with croton oil. Never lose sight of the *hygienic treatment*. Tonics, beef-tea, etc., are never to be neglected. In the last stages you might give stimulants. During the prodromic stage, cod-liver oil and the hypophosphites should be given in all cases.

LECTURE VII.

CEREBRO-SPINAL MENINGITIS.

Anatomical Characters.—Its Nature.—An Essential Fever.—Malignant Scarlet Fever.—Malarial Fevers.—Three Forms: Simple, Fulminant, and Purpuric.—Clinical History.—First Form.—Symptoms—Brain, Spinal, and General: Chills and Fever, Vomiting, Pain, Decubitus.—Second Form.—Third Form.—Re-absorbent Fever.—Death from Asthenia or Coma.—Pathological Anatomy.—Prognosis.—Treatment.—Hygiene.—Morphia for Rachialgia.—Iodide and Bromide of Potassium, Fluid Extract of Ergot, Belladonna, etc.

GENTLEMEN,—In to-night's lecture I propose to treat of a very interesting affection, one which you are liable to encounter at different periods, generally prevailing epidemically, and very rarely sporadically; an exceedingly fatal disease, with which physicians find it difficult to contend, and, if they fully appreciate its nature and are candid, they will readily admit that they dread its occurrence, and have little or no control over its progress. This disease is cerebro-spinal meningitis, sometimes called "spotted fever," also "cerebral typhus," and by many other names,—the consideration of all which would be a waste of time.

Cerebro-spinal meningitis results in an *inflammation of the membranes of the brain and of the spinal cord*. Hence the distinction is easy between this affection and those involving only the membranes of the brain. It is really a cerebral meningitis *plus* a spinal meningitis, the brain and the spinal cord being both enveloped by the same membranes.

As regards its nature, several conflicting opinions exist. Some contend that it is essentially a disease of the nervous system; others, that it is malarial in origin; and still others, that it belongs to the essential fevers. In order to discuss this question fully I would have to devote to it considerable time, and, after all, perhaps would not arrive at a satisfactory solution of the problem. I do not think it a disease of the nervous system, any more than I think typhoid is to be assigned such classification. I believe, with

most neuro-pathologists, that it is the result of a blood-poison,—actually an essential fever, whose whole force and violence are expended upon the nervous system, not unlike the *materies morbi* existing in typhoid fever, which attacks Peyer's glands, causing prominence of the abdominal symptoms, because the principal outburst of the storm is spent upon those organs. Hence, as we do not know the actual character of typhoid fever, it might be better, with Dr. Wood, to call it an enteric fever. It is highly probable that just as a particular *materies morbi* produces enteric fever, so does the blood-poison in cerebro-spinal meningitis induce inflammation of the cerebro-spinal coverings.

Cerebro-spinal meningitis has been held by some to be merely a variety of malignant scarlet fever. All I have to say in this connection is that the theory is too absurd to need refutation, as almost all authors agree that cerebro-spinal meningitis possesses nothing in common with the exanthematous or eruptive fevers. The reason for my not believing it to have anything in common with malarial fever is, that quinine, which possesses such remarkably specific powers in, and is the best and most reliable of all antidotes to, malarial toxæmia, is utterly powerless to arrest or even influence its course. I have given it myself, in large and bold doses, and, notwithstanding my firm belief in its efficacy and extraordinary powers in most febrile complaints, I must confess its total failure in my hands to control this disease in the slightest degree; and almost all other medicines with which I have tried to check the devastations of this dreadful scourge have likewise produced little or no satisfactory results. In reference to the question of its being a kind of "cerebral typhus," I can safely assert that cerebro-spinal meningitis has, in my opinion, no relationship whatever with typhus. There may be, it is true, a similarity between some of the clinical phenomena of both diseases; yet in reviewing the history we must come to the conclusion that cerebro-spinal meningitis is *not* a form of typhus fever. Typhus is a very contagious disease: far more so than cerebro-spinal meningitis. Neither is typhus a disease of such short duration, nor does it uniformly involve the cerebro-spinal meninges. In the cerebro-spinal form of meningitis there are marked symptoms of spinal origin, although there are also purely intercranial ones; but the former are never present in typhus.

Neither is there any similarity between the eruptions of the two diseases. Cerebro-spinal meningitis is, consequently, in all probability a disease *sui generis*, and should be grouped with the essential fevers.

You will perhaps ask yourselves, If this be the case, why speak of it in connection with diseases of the nervous system? I simply do so as a matter of convenience. Some of its pathognomonic symptoms are entirely referable to the nervous system. I thought it proper, therefore, to speak of it at the present moment, in order to differentiate the disease from other forms of meningitis; my chief idea being to give you a proper conception of its peculiarities.

We must next consider the principal symptoms of this affection. As regards their classification, I believe, with Russell Reynolds, that there are three forms or varieties of cerebro-spinal meningitis. I shall not describe them at length, but shall merely review their principal features. The first is the *simple or ordinary*; the second, the *fulminant or siderant*; and the third, the *purpuric* variety.

The ordinary as well as the other varieties rarely occurs sporadically, the disease being generally epidemic, and often devastating whole communities. It occurs mainly in crowded, unhealthy, ill-ventilated places, or where hygienic requirements are neglected. This often happens in prisons, camps, work-houses, and hospitals. There is perhaps a short period of invasion, marked by general *malaise*, and, in abortive cases, by headache and constipation; but I believe the disease rarely aborts, and the patient but too often dies. The mortality is always greatest during the early part of the prevalence of the epidemic. This is usually the rule under such circumstances, the greatest malignancy generally occurring in the earlier cases, and thus spending itself; and, no matter how great your care or how unceasing and earnest your efforts, they will at this period usually prove futile. During its prevalence you will be on the alert: as during epidemic visitations of variola, when everybody grows alarmed at the appearance of no matter what kind of an eruption, and backache; or as in scarlatina, when the slightest throat-trouble causes the greatest anxiety; so during the existence in the community of cerebro-spinal meningitis, the least pain in the occipital region gives occasion for the most serious apprehension.

The first form is the simple or ordinary. The phenomena of the first or simple form of cerebro-spinal meningitis are first *chill*, which is followed by *fever*; then more or less *vomiting*, accompanied by violent *pain* in the *head*, *nucha*, and *back*; also *delirium*.

In order to recognize an acquaintance, we ordinarily think of or recall his peculiar appearance and general conformation of features, not the particular form or shape of any single lineament of his countenance, but their appropriate combination, which characterizes his familiar physiognomy. In this manner we also remember and recognize the individual characteristics of countenances pertaining to other persons. So it is in diseases: the peculiarities of which are not always represented by one symptom, but by a concurrence of many, and also their peculiar grouping and arrangement. So in cerebro-spinal meningitis, we consider, first, the *epidemic prevalence of the disease*; then the *initiatory chill and fever*; the *vomiting, delirium*; *pain* in the *nape of the neck* and in the *spinal region*; besides general cutaneous hyperæsthesia.

Vomiting is always a marked, persistent, and obstinate symptom. The pain in the nucha is violent and enduring. One of the important peculiarities of the clinical phenomena is, that the pain in the spinal region is intensely severe. It is lancinating and darting to the four extremities of the body, its violence being greatly aggravated by the least movement; and hence the decubitus of the patient is very peculiar, and sometimes causes the meningitis to be mistaken for rheumatism, especially as the hyperæsthesia may be misinterpreted.

In cerebro-spinal meningitis you have brain-symptoms, the meninges of the brain being involved; and you therefore find the violent pain in the head, with the existence of prominent delirium. Among the other brain-symptoms are vomiting, insomnia, constipation, contraction of the pupils, which afterwards dilate, and occipital pain. The spinal symptoms are the lancinating lumbar and sacral pains, aggravated by every movement, and accompanied by more or less spasm, mostly of the muscles of the neck, thus fixing the head backwards on the spine. In some cases the spasmodic contraction is so extensive as to draw the whole body violently and firmly backwards, producing *opisthotonos*: generally the spasm is limited to the nuchal region.

The general symptoms are fever, with a temperature varying

from $103^{\circ}\text{--}4^{\circ}$ to $106^{\circ}\text{--}7^{\circ}$ F.; more or less constipation, sometimes diarrhoea, anorexia, prostration of the vital powers, due to the implication of the nervous centres, and insomnia, or, in some cases, stupor. There is often an herpetic eruption in the neighborhood of the lips, and an ecchymosis on the body, giving the disease the name of "spotted fever." These symptoms constitute the ordinary form of cerebro-spinal meningitis. Of course they vary with different epidemics; and so at times the eruption will be greater or less, or even absent; the disease also differing in violence, intensity, and duration.

The second form is the fulminant or siderant form. This means the thundering form, and in truth it is not only thundering but also lethal. Its malignancy is so great that it has proved fatal in five hours. If a blood-poison is of sufficient virulence to produce such terrible results, what can a physician accomplish in his efforts to oppose its progress? He can achieve nothing. What compose the peculiarities of this form? Have you ever seen the stage of collapse of epidemic cholera, the algid state, as it is called? If you have, you will remember the ghastly pallor, the shrunken condition and clammy coldness of the skin; the pulselessness and general prostration; with the cyanotic appearance of the mucous membranes, and rapid sinking of the vital powers. These phenomena are also seen in the fulminant form of cerebro-spinal meningitis, with apoplectic phenomena in addition. You thus have the algid state of cholera, *plus* coma. There is little possibility of reaction, and at your first visit your patient is moribund; at the second, dead.

The third form is the purpuric. This form occurs as a combination either of the first and the second, or of the first with purpuric symptoms, in consequence of extensive and profound blood-poisoning, the result of dyscrasia or true necræmia. There is a tendency to extravasation of blood [in the subcutaneous and submucous tissues; the capillaries, being friable from want of tonicity, rupture; hemorrhage follows, and petechiæ appear. No matter what combination of symptoms accompany this variety, there is an intense, malignant blood-poisoning, a necræmia, and a conjoint appearance of spots and blotches of a purpuric hue upon the surface of the body. There is another remarkable fact in regard to the symptomatology of this disease, referred to by Ziemssen.

Those patients who do not die during the first or second week of the attack are not entirely free from danger. Very often a fever is developed, called the "re-absorbent fever," which is a pyæmic condition, induced by the absorption of peccant matter. In this disease, as well as in simple cerebral meningitis, a sero-purulent exudation collects in the subarachnoidean spaces, and at the base of the brain, causing an impairment in the functions of the nerves of special sense.

After the primary fever has disappeared, the re-absorbent fever develops during convalescence. An absorption of the exudations occurs; and if the resulting pyæmia be not fatal, the patient evinces signs of commencing recovery, though still having one more danger to incur, which is marasmus, in character not unlike *tabes mesenterica*. The nervous centres, presiding over the functions of nutrition, are probably at fault, damaged by the violence of the acute stage; a pernicious diarrhoea and progressive wasting soon reduce the patient, in spite of beef-tea, wine, and tonics, to a mere skeleton. Finally, death by asthenia occurs; although at earlier periods of the disease it may be from coma.

As we have considered pretty thoroughly the nature, as well as the etiology, of this malady, little remains to be said of its pathology or pathological anatomy. We know that cerebro-spinal meningitis is primarily a disease of the blood, with inflammation of the membranes of the cerebrum and medulla spinalis, resulting from toxæmia, and attended by an effusion of serum, lymph, and pus. If death is produced by necræmia, no trace of meningeal inflammation is found, because there has not been sufficient time for the inflammatory condition to produce the transudation.

Considering all that has been said, you cannot be ignorant in regard to the prognosis. It is of very grave import, and experienced physicians fear to encounter this dread disease. I hesitate to consider its treatment, having tried almost everything with but slight beneficial result. I have lost many cases, some dying in spite of every effort to save them. Others recover almost unaccountably. Of course, upon reference to your books you will find modes of treatment *ad infinitum*; but test their efficiency at the bedside, and observe the results, and your faith in medication will probably be completely shattered. Try for yourselves, and profit by your own experience. I doubt if we

will ever be able to make great progress in mastering epidemics. In all cases be guided by your experience, your convictions, and the earnest desire to achieve your utmost. I do not wish to trammel your memories with a catalogue of therapeutical resources, as I have found no satisfactory result from any medicaments in the cases which have fallen under my observation. Keep up nutrition and sustain the vital powers. During rachialgia, you may give morphia hypodermically, taking care not to give too large a dose. You might administer iodide and bromide of potassium and fluid extract of ergot; the two latter, by acting on the vaso-motor nerves, may control the hyperæmia of the meninges. Belladonna, cannabis Indica, quinine, counter-irritants, etc., have all been recommended. I have only to add, in regard to remedies, that their number is generally in direct proportion to the hopelessness of the affection. Their multiplicity corresponds with their inefficiency.

LECTURE VIII.

PACHY-MENINGITIS.

Forms of Meningitis.—Pachy-Meningitis.—Etiology: Blows, Injuries, Ozæna, Otorrhœa.—Anatomical Characters.—Clinical History.—Inflammation of Cerebral Sinuses.—Thrombosis.—Metastatic Abscesses in the Lung.—Symptoms.—Causes of Death.—Treatment.—Prognosis.—Pathological Anatomy.

GENTLEMEN,—In my last lecture, while speaking to you upon the subject of cerebro-spinal meningitis, I discussed its connection with different diseases, such as typhus fever, scarlatina, malarial fever, etc., with which, at least by some authors, it has been confounded. I fully compared its clinical and pathological phenomena with those of each of these, proving conclusively that it was not in any manner connected with them. I, moreover, took the position that it was not primarily a nervous affection, but an essential fever, somewhat resembling typhus in its action, and that in cerebro-spinal meningitis a peculiar *materies morbi* probably existed, whose action upon the cerebro-spinal nervous system caused the inflammation of the meninges, there being a toxæmia in both cases.

In the consideration of the different diseases of the membranes covering the brain we have so far reviewed: first, acute idiopathic; second, tuberculous; third, cerebro-spinal meningitis. In each of these diseases there is, as we have already seen, an inflammation of the pia mater as well as of the arachnoid. We now come to the description of the fourth form, the last one we will study. It is a variety of rare occurrence, but in reference to which, however, you must be constantly on your guard, never allowing it to elude your vigilance, as it is apt to deceive the inexperienced physician. This affection is known as *pachy-meningitis*, or inflammation of the dura mater.

If you have carefully followed me in my previous lectures, you will recollect that in the other varieties of meningitis the dura mater was not involved. In the present malady, however, the

inflammation is almost exclusively limited to that membrane, the others remaining healthy.

Pachy-meningitis is rarely idiopathic, being almost always dependent upon some secondary cause; hence, when it exists, we can generally suspect the nature of its etiology. It differs considerably in this respect from acute idiopathic meningitis: a child, for instance, is often seized with the latter affection, without our being in the slightest degree able to ascertain the exciting cause; but in pachy-meningitis there are certain generally-recognized influences leading to its production. The most ordinary of these are *severe blows upon the head*, and *external violence, fractures or fissures in the skull*. In addition to these, we have diseases of the bones of the cranium, such as *caries* (syphilitic or otherwise), resulting from *ozæna*.

Another very common and important source of pachy-meningitis, which I wish you always to recollect, and one which is not sufficiently appreciated or recognized, is *otorrhœa*, with *caries* of the temporal bones. *Otorrhœa* is a frequent sequel to *scarlatina*, or other of the *exanthemata*, such as *rubeola* or *variola*. In these diseases there is usually an affection of the throat, an inflammation of the pharynx, which is more or less persistent, and, being situated in the mucous membrane of the fauces, by continuity of structure is transmitted along the mucous membrane of the Eustachian tube, and finally involves the middle and internal ear. This rapidly destroys the *ossicula auris*, attacks next the deeper layers of bone, and finally produces an inflammation of the *dura mater*, developing pachy-meningitis, as we have before seen. Jaccoud states that when the disorder follows an injury, as a blow upon the head, the starting-point of the inflammation is in the membrane lining the external surface of the skull,—the *pericranium*. The inflammatory condition of the *pericranium*, for reasons not obvious, causes the inflammation of the *dura mater*: as there is no very evident connection between these membranes, we cannot very well explain the mode of transmission of the inflammation; though, after all, it might be communicated or propagated through the osseous structure.

Considering what I have said in regard to *otorrhœa*, you will readily understand the necessity and importance of energetically treating the disease, notwithstanding the representations of parents

that its cure is attended with danger. As a rule, mothers do not wish an interference with any discharge. This is an old-fashioned but still prevalent prejudice. Formerly it was considered very injudicious on the part of a physician to arrest or check purulent discharges. I have seen little children, covered with eczema, scratch, suffer, and pass sleepless nights, simply because the family physician acquiesced in the wishes of the mother, who, according to some traditional notion, imagined that brain-disease would inevitably follow the disappearance of the eruption. These ideas, as I have said already, are held not only in regard to otorrhœa, but also to cutaneous eruptions, having some authoritative weight in their support. Owing to such opinions, many an otorrhœa has been allowed to run its pernicious course, causing caries of the neighboring bone, inflammation of the dura mater, and the death of the patient. It is always well to respect the feelings of the mother, but you should never allow yourselves to be dictated to by any one governed by prejudice. Rather decline the responsibility of the case.

In order to show you the suddenness of death in some such cases, I will relate an incident to you which came under my direct observation. Before commencing its recital, I can conceive that you may perhaps inquire if in pachy-meningitis we have not first the symptoms of irritation and then of depression, or if there will be marked headache, convulsions, vomiting, contraction of the pupils, etc., previous to the advent of coma. In answer, I would say that in some cases they may be present, in others they may be absent; and oftentimes the first symptomatic indications will be those of depression, those of irritation having been so slight that they were entirely overlooked, and coma will follow. But I must relate my case, as an illustration from actual experience is always much more instructive than a long disquisition. I wish particularly to impress you with caution, by citing to you not my triumphs but my mistakes, in order that you may be prevented from being led into the same error.

Some years ago I was the family physician of a most respected and interesting family, one of whose members was a young lady of about the age of eighteen. She was a charming girl, very intelligent and highly accomplished, and had had during childhood an attack of scarlatina, followed by an otorrhœa so obstinate and

persistent as to defy all treatment. A distinguished specialist in aural surgery had treated her without success. About the time of the sad occurrence I am relating, the young lady was noticed to be failing in health, which was all the history I could glean. She was not very sick, but the mother had become uneasy and sent for me. I also learned that there had been some fever, and, being somewhat in a hurry, I diagnosticated rapidly an intermittent fever which was then quite prevalent. Upon questioning the mother further, I learned that the girl had had severe headache for a few days previously, and also that she imagined her daughter was at times somewhat delirious. This was perfectly compatible with my diagnosis. As she also had a sore throat, I proceeded to examine it carefully. Bringing her near the gas-light for the purpose, I noticed that the light greatly hurt her eyes, but paid no attention to this important fact; still believing that she had malarial fever, and her tongue being coated, I prescribed calomel and quinine, and then left, promising to return the next day. The mother, being nervous and anxious about her child, followed me to the door and asked for my opinion. I immediately proceeded to reassure her, firmly believing that the quinine would do its work, so I told her that there was no cause for alarm, as the young lady would be well in a few days. About eleven o'clock the same night I received a message from a neighboring physician, who desired my presence at the house of my patient, stating also that she was dying. I thought it was probably some hysterical trouble, making him over-anxious, but still went, intending to reassure him. I had scarcely entered the room before I recognized that she was comatose, and the same minute I appreciated my sad error in diagnosis. I had overlooked the importance of the otorrhœa, although aware of its existence, which, to quote the beautiful expression of Niemeyer, is like the "sword of Damocles, suspended by a slender thread." The otorrhœa, photophobia, headache, constipation, and delirium were all known to me at my first visit, yet I overlooked the danger and gave an encouraging prognosis! I was baffled and mortified, as the lady died that same night, and I should have anticipated the unfortunate termination of her illness. It was a lesson which is still indelibly impressed upon my mind. If this mistake of mine can be at all beneficial to you, if an otorrhœa with cerebral symptoms can make you

apprehensive, and sound the note of alarm when presented for your consideration, then I am amply repaid in having related my melancholy experience. You will have remarked how very few were the symptoms of irritation in this case, how rapidly coma supervened, carrying the patient off before alarming symptoms had manifested themselves. You see, therefore, that pachy-meningitis is an affection to be dreaded. This case illustrates the mode of extension from the ear to the dura mater, resulting fatally.

You are all acquainted with the peculiarities of the dura mater, its sinuses, and their peculiar anatomical relations. Now, when an inflammatory condition of the dura mater exists, there will be developed a tendency to the formation of thrombi in the cerebral sinuses. The inflammation of the dura mater may be propagated to the sinuses, stasis of blood will occur within them, and a clot or thrombus being formed will interrupt the circulation and clog their cavity. This is one of the contingent dangers of inflammation of the dura mater, and according to the location of the primary cause will a particular sinus become involved. In ozæna and caries of the ethmoid bone the longitudinal sinuses will be implicated, while in caries of the petrous portion of the temporal bone the lateral and petrosal sinuses will be inflamed.

From your knowledge of thrombosis and embolism, you are aware that the interference with the circulation in the cerebral sinuses is not the only danger to be apprehended, as there may be another important complication,—a metastatic abscess in the lung. You should always remember that thrombosis may result in embolism, as sometimes occurs, for instance, after inflammation of the uterine sinuses, and also in phlebitis resulting from fractures or other causes. We have already seen how the embolus becomes detached, and is taken to the right ventricle and thence to the lungs, where, if large enough, it will plug up the pulmonary artery, or one of its important branches, producing death by apnœa. But if the clot be small and in a suppurative stage, a metastatic abscess will be produced in the lung. This is exactly what sometimes happens in pachy-meningitis. The notions upon this subject were, up to a recent date, of a very crude character. You will now be able fully to realize the danger of this disorder, and also to understand its mode of origin.

Unfortunately, there are no particular or pathognomonic symptoms of this affection. You should, however, be constantly on your guard as to the existence of the conditions of the primary disturbance, which, with the history, will give you a clue to the diagnosis. If the patient has received a violent blow upon the head, if he has otorrhœa or ozæna of long standing, and before death exhibits marked cerebral symptoms, you may safely conclude that the disease is probably pachy-meningitis. The symptomatology of the affection may be obscure, but the etiology remains clear. Hence it is that I do not wish to dwell upon unimportant symptoms, the main object being that you should be fully acquainted with the causes of the disease, and that thus being forewarned you may be forearmed. Never hurry in making a diagnosis, and always attach paramount importance to otorrhœa and ozæna. These you should treat in time to prevent subsequent symptoms that might arise, otherwise coma will supervene, and you will be utterly powerless to effect any good. To recapitulate: recollect that in otorrhœa, ozæna, and injuries to the skull, you are to apprehend pachy-meningitis, and that as a result there may be thrombosis of the cerebral sinuses. The patient may die of arrestation of the circulation in the sinuses, of inflammation of the dura mater itself, or of embolism or its result,—metastatic abscess of the lung.

About the treatment there is very little to be said. You may treat the brain-symptoms in this disease as in other forms of meningitis.

The prognosis, of course, is necessarily very unfavorable. On post-mortem examination you will generally find an adhesion of the dura mater to the calvarium. Sometimes, but very rarely, the other membranes are involved. There is a hardening and a thickening of the dura mater, and a purulent deposit between the membrane and the bone. You find, moreover, an inflammatory condition of the cerebral sinuses, thrombi or clots, large or small, and at times markedly putrescent,—sometimes extending as far as the torcular Herophili.

LECTURE IX.

INSANITY.

Insanity a Disease of the Brain.—Its Origin.—Location.—Predisposition.—Insanity hereditary.—Definition.—Unconscious Cerebration.—Moral Insanity: Examples.—Rules for ascertaining Insanity.

GENTLEMEN,—I propose to-night to discuss the subject of insanity. No one will say that this is an exceedingly rare disease, and hence not worth our serious consideration. This would be an erroneous conclusion in even the most exceptional forms of ailment, because when you start out practicing you must expect and be prepared to meet all disorders. Insanity is of frequent occurrence. It is not a disease of the mind, but a peculiar variety of affection of the brain, and one which, unfortunately, you will but too often meet. You will undoubtedly be called upon to express your views in regard to its nature, and will naturally be expected to possess some knowledge of its leading features. Even in its general outlines insanity is a subject belonging to the practitioner, being as much a disease of the brain as any other affection of that organ. It consequently claims his attention as much as any ailment of any other portion of the human body. Though a true disease of the brain, it does not invariably have its origin there; indeed, it may start almost anywhere else, as in the diseased uterus in the female, or in some morbid condition of the colon, and in either case it will disappear upon proper treatment being addressed to its source, thereby removing the cause.

When a man is insane there is always a disturbance of the normal working powers of the brain. Insanity may have the greatest multiplicity of primary causes. In the majority of cases, I believe, insanity originates at a greater or less distance from the brain, and hence it is mostly a secondary affection, being rarely idiopathic.

We have now made one step towards the proper elucidation of this subject, in having established that insanity is not necessarily

a stigma upon a person, as was formerly supposed; nor is it, metaphysically speaking, a disorder of the mind, with accompanying mental disturbances not easily understood. It is an affection of the brain, which, though not always originating in that organ, invariably has its seat there.

You will not find it difficult to appreciate these facts if you recollect that the brain is the supreme centre, presiding over all the other parts of the nervous system and of the animal economy. Indeed, we can hardly conceive of any constituent atom of the body, no matter where found, which is not in more or less intimate relation with the brain. It presides over multitudinous atoms, its influence is felt throughout the body, and there is not a muscle, nerve, artery, or vein, not even the smallest histological element in the human system, but is permeated by the peculiar nervous force or influence emanating from the "supreme cerebral ganglia." How easy, then, is it to understand that the functions of the brain may be more or less impaired by the presence of disease, especially where there exists any predisposition to insanity!

In all disorders leading to insanity this predisposition undoubtedly exercises great influence and plays a most important *rôle*. That it is inherent in many instances there can be no doubt. In insanity, as in other maladies, a great many facts are manifest which we would not be able to interpret but for the assumption of predisposition or heredity. Why should one member of a family die of phthisis, and another, placed in the same conditions, be unaffected? Or, in scarlatina, independently of its contagious elements, why is it that one child, though unprotected, may be exposed with impunity, and another will contract the malady in its malignant form and thereby perish? Or, again, why is it that after vaccination one person may never again be susceptible to the virus, and another one will be re-vaccinated almost at pleasure, and vaccinia readily reproduced? In re-vaccinating persons in large communities, I have several times been struck by the fact that the operation was often successful in those who had been inoculated "in the old country" or were distinctly pitted with pock-marks from varioloid. Why should this be? It is probably on account of a marked predisposition existing in the system. Why should one person be effectually protected by one attack of small-pox and another one die of the third, as happened in London in

a case related by Dr. Watson? This is again undoubtedly due to the cause just explained. So it is in insanity. Some people are very much inclined to it, and labor under a very unstable condition of the nervous system, the normal equilibrium of which is easily disturbed. In many cases, such being the order of events, the patient invariably goes mad; while another person, subjected to exactly the same influences and conditions, will not experience the slightest variation in the performance of his intellectual functions.

Upon inquiry into the history of the insane, you will generally find that the predisposition does not originate with the individual, but existed prior to himself; that is, it is mostly hereditary and transmitted, and this tendency to insanity is an heirloom. You often hear insanity spoken of as a mental aberration, lunacy, a condition of *non compos mentis*, etc.; but insanity is the best term of them all. Insane literally means deprived of reason,—not in a normal state of mental health,—unsound in mind. Now, can there be given an exact definition of insanity? Can a line between insanity and reason be drawn so definitely that all the phenomena found on one side shall be compatible with reason, and those on the other with insanity? This is impossible, and, as a great author says, “The shades of variation in eccentricity, between sanity and insanity, are so slight and numerous that it is exceedingly difficult to state, where reason ends and insanity begins.” This you might perhaps have had occasion to exemplify, as you may have a friend in whom at times you have noticed very strange actions. Sometimes you will think that if you were not acquainted with the man you would suppose him to be crazy, or not be surprised if some day he should lose his mind. This would be the result of noticing actions in him occasionally not apparently compatible with an integrity of the mental faculties; and then his deportment at other times may entirely disprove your apprehensions. Such people may be said to live upon the border-land of insanity, and are very apt occasionally to cross the line and return; occasionally they are crazy, at other times they are not. Learned and eminent men “cross the line” sometimes, and make short excursions into the labyrinths of intellectual aberration. At all events, there is in some people a peculiar predisposition to get over the line, and their peregrinations and rapid transitions mystify medical experts.

No matter how much experience you may possess, you will occasionally be unable to decide the status of such cases. It may in certain circumstances, fortunately rare, take a great length of time. Close observation day after day, week after week, even month after month, will be requisite before you may be able to arrive at a safe and definite conclusion, and occasionally you may be baffled even then. This may seem very strange at first thought, but upon studying the various phenomena and difficulties of the subject it will not appear so remarkable.

Did you ever see two persons who exactly resembled each other, and between whom not the slightest difference existed in features, countenance, or appearance? Even the voice presents a dissimilarity in different persons, being coarse and harsh in some, soft and melodious in others. Understand, that as faces and voices differ, so are the mental characteristics distinct. Again, if persons vary so much in their physical and mental capacities and qualities in health, how much more will they disagree when affected by disease! Besides this, great changes and varieties of development are produced in sane minds under different influences and circumstances. The mental faculties become blunted by neglect of education, or they may be constantly improved and developed by mental gymnastics. In men who habitually study and think there is also, figuratively speaking, considerable mental absorption constantly occurring, by what Dr. Carpenter terms "unconscious cerebration." Some authors contend that the most brilliant thoughts often result from mental activity of this type. Have you never involuntarily thought of something while your attention was engaged in pursuing other subjects, and, being struck by the pertinency of the thought, immediately noted it, in order to retain it in your memory? Or have you never retired, after assiduously striving to master a perplexing subject, at last abandoning it in despair, seeking repose to recuperate your energies for renewed efforts, and awaking the next morning after a sound sleep clear-headed and bright, to find the chaotic confusion gone and yourself in perfect possession of that which a few hours before you had despaired of ever attaining? This is "unconscious cerebration." You were appropriating the knowledge in spite of yourself, and after the invigorating rest the mind manifested what you had so thoroughly, without perceiving it, acquired. It is

through the channels of the senses that man is educated, by a perception effected in the cerebral convolutions of whatever the senses may have transmitted; which perception always involves a working of the cortical cells, and in proportion as we cultivate their working power do we add to their capacity. As the mind is capable of cultivation, so are the individual senses; and, by the laws of compensation, one sense may supply a lost one, and a blind man's hearing and touch may become so preternaturally developed as to be truly marvelous. But suppose a man to have arrived at the age of thirty without ever having enjoyed the use of his senses, and then all at once they be fully developed, what would be the result? He would be an idiot; never having been able to cultivate them, he could not appreciate their working, and time must necessarily elapse before he could become benefited therefrom. Now, what does all this digression signify? Simply that I cannot give a distinct definition of insanity that would be comprehensive, because of its protean character; on account of the great and almost infinite dissimilarity existing between the minds of different individuals, which will be more distinct when pathologically disturbed. Insanity in one person may produce actions not unlike those of the brute; while in another it may lead to actions of a very different character. So that there can be no constant, pathognomonic phenomena which may be said to be attendant upon mental alienation. Of course we have certain classifications, but some of the manifestations in insanity will not admit of any special grouping. I believe that upon the witness-stand you may prudently decline to attempt any definition of insanity, for reasons just mentioned.

"Insanity is," according to Shephard, "a disease of the neurine batteries of the brain." This definition has at least one merit,—it will puzzle the lawyers. It sometimes happens that when medical evidence is required in court, the legal gentlemen, being very shrewd, possessing a knowledge of medical jurisprudence and perhaps some smattering of medical science, seek definitions for their own purposes, and will afterwards endeavor to entangle you in their mazes. For these very considerations, the more concise and explicit you are in your testimony, the better. Another definition is that of Maudsley, who states that "insanity is a morbid derangement, generally chronic, of the supreme cerebral

centres, the gray matter of the convolutions, or the *intellectorium commune*, giving rise to perverted feeling, defective or erroneous ideation, and discordant conduct, conjointly or separately, and more or less incapacitating the individual for his due social relations." Now, in some respects, this definition is deficient; it is not sufficiently comprehensive, possessing some flaws, and not covering all cases. Still, it has undoubted advantages, because it states—first, where the disease is located, and, secondly, that the disease is usually chronic. According to Van der Kolk, all insanity is acute which has not existed over three months. We are not to infer from Maudsley's reference to the fact of the ailment being usually chronic that it was not originally acute, but simply that a physician is, as a rule, called to treat it after the condition has become more or less chronic. This division of the different results of insanity is also in accordance with the best division of the mind,—that is, *will*, in relation to those parts giving rise to the phenomena of *action*; *feeling*, in relation to those parts giving rise to the phenomena of *emotions*; and *ideation*, in relation to those parts giving rise to *cognition*.

Hence it follows that a man may be insane in his words, as evidence of a perverted manifestation of intellect; or in his emotions, as expressive of a morbid state of those parts which preside over the development of his feelings; or in his actions, as expressive of disorder of that portion of the brain connected with the phenomena of volition; or there may be insanity exhibited in his actions, emotions, and ideas, conjointly; one, or the combination of two, or all the three forms of deranged manifestations just described, may be present.

Man is often said to suffer from moral insanity, which is claimed to be an affective variety, referring exclusively to the development of irregular emotions, as opposed to the perversion of the purely intellectual faculties,—there being in moral, affective, or pathetic disorders a disturbance of the moral faculties only. In former years I advocated this distinction; but of late I have had reason to modify the views I so long taught, and am now convinced that all cases of moral insanity, closely scrutinized, will present evidences of imperfect ideation, superadded to the derangement of the affective faculties. The point to recollect for the present is, that a person may be insane in one, two, or three forms of mani-

festation. Sometimes it is difficult to determine the existence of insanity, because some individuals possess a remarkable control over themselves, perhaps being designing and deceitful; others, presenting some suspicious symptoms, will talk coherently and intelligently, oftentimes puzzling experts as to their actual psychological status. You see, therefore, that a man need not be a raving maniac in order to be insane; he may be very courteous, intelligent, polished, and affable, and still be hopelessly crazy.

I recollect two interesting cases, which, in this connection, I will relate to you. The first was that of a man subjected to a commission *de lunatico inquirendo*. All the experts who knew him swore that he was insane. But he contended that he knew much more than the doctors did, at least about his own case. He wished to address the jury, was permitted to do so, and made a most brilliant and persuasive speech; after which, of course, the jury declared that he was not insane, and consequently he was permitted to return home. The very first day of his enjoyment of liberty, he choked his wife nearly to death because she refused to drink out of an old skull which he possessed.

The next case was that of a patient to whose mental alienation I swore most emphatically. He grew very angry with me, and even threatened to horsewhip me. He took exceptions to the fact that the court had appointed an attorney to defend him, stating that he was fully able to protect himself; that he entertained the most supreme contempt for lawyers; and, at the commencement of the trial, he ignominiously dismissed his counsel. He conducted his own case quite energetically, examining the witnesses with deliberation and care, seizing the weak points in their answers, and evincing skill and acumen. The jury were evidently staggered by his brilliancy, until one or two preposterous assertions into which he was betrayed convinced every one that his insanity was indubitable. He eventually died of general paresis.

Such cases actually occur; and these illustrations are not in the least overdrawn. You will often have very cunning insane people to deal with. I remember an old judge who was at St. Vincent's, and who was a monomaniac on the subject of perpetual motion, though apparently sane on every other subject. He always appeared to be very intelligent, and, after a long stay in the asylum, I felt convinced that he should be discharged. I

therefore determined to let him go; and the judge, exceedingly thankful and grateful, returned home. The first week after his arrival, he became so violent that he was sent to another asylum; and yet I made this mistake after having had him long under my immediate observation. You have seen here exemplified what you have already learned, that an insane man can successfully conceal his derangement, and, eluding our vigilance, perpetrate some deed of violence. The conversation may show no symptoms of the affection, which will sooner or later declare themselves. If these sources of error exist, is there any particular rule by which we can clearly ascertain the existence of insanity? Unfortunately, there is not; but there are some points of importance which are invaluable, and have always been of great assistance to me in the diagnosis of difficult cases.

In the first place, when a man is insane, he is "*out of harmony with his social relations*," which makes him apparently unfitted for his accustomed sphere of life. There is, on his part, a want of adaptation to, and congruity with, his surroundings, which is quite evident, and a certain inconsistency of thought, action, or feeling irreconcilable with the idea of sanity. In the next place, and this is a universal rule, insanity consists in an invariable *departure from one's normal self*. To illustrate what I mean by this change and impress it upon your minds, we will suppose that after this lecture some gentleman of this class were suddenly to jump down here and execute a series of fantastic acrobatic feats. Such an action on his part would not necessarily be presumptive of insanity. He might be in the habit of performing such tricks and antics, being uncommonly vivacious in disposition. But if our venerable Dean were to go through the same manœuvres, considering his advanced age and his natural dignity of character, we would not then have to seek for further evidence of madness. Suppose, on the other hand, that suddenly a chaste, refined, temperate, studious man, contrary to his habits and former inclinations, were to get drunk often, use obscene and loathsome language, associate with prostitutes, show a great disturbance and change in his moral qualities, would you not tell his wife to beware, that he was going mad, and, if not watched, he might commit some overt act of violence? But if a man were an habitual drunkard, and a regular visitor of brothels, etc., you surely would not concern your-

self about his habits. Hence you will understand what is meant by a departure from one's normal self; and, in order to be able to judge, you must be acquainted with the previous character and history of the patient. If this departure be once established in your mind, your case is made out. Another means we have of comparing his actions, is what is called the *sensus communis* of persons. There are established in society certain common rules of deportment, which people observe, and a departure from them by one who has always conformed to them will often help the diagnosis, especially if the deviation consists in acts conflicting with this established sense of propriety. The person, then, being no longer a "unit in the social harmony which should prevail," can be considered crazy or insane, and should be isolated until restored to mental health and usefulness: otherwise he might irreparably injure himself and endanger others.

LECTURE X.

INSANITY—*continued*.

Classifications.—Maudsley's Classification.—Etiology.—Predisposing Causes: Climate, Religion, Civilization, Sex, Period of Life, Deficient Education, Individual Predisposition, Insane Temperament.—**Exciting Causes:** Masturbation, Drunkenness, Epilepsy, Transmutation of Nervous Diseases, Chronic Diseases, Disorders of the Sexual Functions, Injuries to the Head.—**Moral Cause.**

GENTLEMEN,—In my last lecture, when speaking of insanity, I alluded to important facts regarding its character. It now remains for me to *classify* this disease in accordance with the views of the best authorities. It is quite as difficult a matter to classify insanity as to define it, and there are instances where it is impracticable to arrange some of its manifestations typically.

I am a believer in the simplest possible division of the different varieties of this disease, in order not to overburden the mind, and am convinced that Pinel's method is most satisfactory, being as follows: 1st, *Mania*; 2d, *Melancholia*; 3d, *Dementia*; 4th, *Imbecility* and *Idiocy*. (The last two affections are simply for practical purposes here embraced as one.) Some authors have divided the subject into almost numberless classifications. Dr. Skae has originated a very elaborate one, its probable advantage consisting in the application of the different types according to their etiology.

Van der Kolk, of Utrecht, to whom medical science owes a great debt, deals in two principal forms: 1st, *Idiopathic insanity*, arising without any appreciable cause whatsoever, being primary in its character and originating exclusively in the brain. (You will remember that in my last lecture I explained that insanity is always located in the brain, though sometimes its origin may be from sources altogether foreign to that organ.) 2d, *Sympathetic insanity*, which includes all forms proceeding from different portions of the human economy, as, for instance, the viscera of the thorax, or, more frequently, of the abdomen or pelvis.

You will thus perceive that in sympathetic insanity the origin

may be traced to the heart and lungs, to the liver, spleen, small intestines, colon, etc. Insanity is indeed quite often sympathetic with a diseased colon,—especially where melancholia exists,—the starting-point not infrequently being in some portion of the alimentary canal. Again,—as all practitioners very well know,—certain diseases of the genito-urinary apparatus not rarely produce the most obstinate forms of mental aberration. It is not difficult to understand why diseases of these organs should sometimes have this determination, if you take into consideration the complete harmony normally existing between all parts of the body and the central organ of the nervous system—the brain. No matter how remote their situation, many organic complications give rise to mental disturbances by the reflex irritation excited first in their nerve-fibres and then propagated to the central nervous depôt.

One more fact in connection with the sympathetic form is, that these remote causes, though primarily producing only simple functional disturbances of the brain, will, if not removed,—the reflex irritation persistently continuing,—produce an organic disease, developing an incurable form of insanity. The more obstinate the duration and the more violent the irritation, the greater is the danger of causing organic changes in the histological elements of the brain,—the resulting insanity becoming irremediable.

Another classification which I shall mention is that of Maudsley. It is excellent, and I purpose following it in this course of lectures. He divides insanity into—1st, *Mania*, acute and chronic; 2d, *Monomania*; 3d, *Melancholia*; 4th, *Moral Insanity*; 5th, *Dementia*; 6th, *Idiocy* and *Imbecility*; and 7th, *General Paralysis of the Insane*.

The next time we meet I shall give a short description of these forms. Contenting ourselves for the present with this brief allusion to the classification adopted, we next shall consider the causation, or the *etiology of insanity*.

Insanity is not, as was formerly supposed, a mere hypothetical disease,—an imaginary or metaphysical affection of the mind. The mind, being intangible and immaterial, cannot be subjected to disease. Insanity is a disorder of the *brain*, just as pneumonitis is an inflammation of the lungs, pleuritis an inflammation of the pleural sac, or typhoid fever a specific blood-poisoning; and the disordered evolution of mental phenomena observed during the

course of insanity is but the morbid manifestation of a brain which, from physical causes, originating in disease, is unable to perform its proper physiological functions.

The fact that insanity is a disease of the brain is one of the greatest importance, showing us the gross injustice inflicted in times past upon individuals, casting upon them, as it did, the stigma of social ostracism. It was supposed that they were suffering from some uncommon malady wrought by mysterious influences, or sent as a punishment from the gods. Such theories were as preposterous as they were unfortunate. All individuals are more or less liable to this calamity. But why are some people insane and others not? Why is not insanity contagious? Why does it not affect indiscriminately all the members of a family? Why does it show a predilection for certain persons, selecting its victims with remarkable consistency? We have already studied the laws relating to this liability, and have established the fact that it is due to an *unstable condition of the nervous elements*, peculiarly prone to be perturbed in their equilibrium, which perturbation produces more or less interference with all the functions connected with the development of mental phenomena; and the person thus suffering will cease to be rational and responsible. Where such a predisposition exists there is likewise an unstable constitution of the cells connected with the evolution of the moral or intellectual faculties; and this instability is born with the individual and inherent to him, having existed from the first moment of his life. Let us suppose two individuals, one of whom is subjected to violent mental emotions, or to other causes eminently calculated to lead to insanity. Should no predisposition exist, this person will pass through the ordeal unscathed. The second individual, on the other hand, possessing the liability, on being exposed to precisely the same agencies, will most assuredly become mad,—the cortical cells now no longer performing their duty normally.

If we knew all the causes leading to insanity, or could foresee contingencies which may arise, then we might venture to predict insanity in certain individuals or families at particular periods of their lives. However, insanity does not depend upon one, or a few, but upon a *multiplicity of causes*; and, as Maudsley remarks, “hereditary, predisposing moral and physical causes are not alone

necessary, but a combination, a concurrence of conditions, and then lunacy follows."

I will now present some of the leading causes of insanity.

1st. *Climate*—which seems to have some influence in the production of this malady. Indeed, I have frequently observed that on dark, moist, gloomy days the majority of insane persons are worse: the melancholic cases are more deeply depressed, and the maniacal more difficult to control. There seems to be also a peculiar disposition towards a cure at certain periods of the year,—in spring especially, when everything in nature is being endowed with renewed life and vigor. In curable patients we then frequently find the disease singularly yielding,—a fact mentioned by Esquirol. Most of you have probably experienced a peculiarly depressing effect upon a gloomy day when the sky is overcast. And if this influence be felt by sane persons, how much more will it impress the insane, or those in whom an hereditary predisposition exists! Should this influence be unduly continued in these predisposed persons, insanity will be developed; which fact partly explains why suicides are so frequently committed during dull and dreary weather.

2d. *Religion*, it is held, is often a cause of insanity. In nervous temperaments subjected to intense religious excitement, as revivals, for instance, the dangers of mental diseases are to be apprehended. I have witnessed many instances of this kind, disastrous nervous complications having fanaticism as their immediate precursor. Indeed, the effects of the mental emotions, reacting upon the physical organism, are so extraordinary, owing to their expansive character, that it is easy enough to understand how they may be fruitful sources of insanity. Maudsley aptly remarks, in his work on "Body and Mind," that when the emotions are very much disturbed there is a proportionate disarrangement in the cortical cells presiding over the moral functions, nervous influence being liberated with great force; this is transmitted along certain appropriate nerves, and the impulse consequent upon the original liberation of nerve-force may be so great as to produce intense and notable effects at distant points, where it is received and ultimately distributed. These phenomena can best be compared to those produced by a powerful galvanic battery, generating a current of great tension, transmitted along conducting wires; a violent shock

occurring at the point of arrest; and this point of arrestation furnishing a resistance corresponding with the strength of the discharge, destructive and disorganizing effects will ensue. This is not unlike what occurs when a person is struck by lightning. Agitation, from either intense grief or unbounded joy, may be so excessive as to produce sudden death. Death thus occasioned was formerly commonly supposed to be the result of a broken heart; but in reality it is simply the effect of the overpowering violence of the emotions upon the nervous centres, and not, as was previously imagined, a muscular laceration.

3d. Another predisposing cause is said to be—*civilization and its progress*. It is true that among savages insanity is exceedingly rare. In what way does civilization tend to produce insanity? you ask. Perhaps by the expansive wants therefrom arising, and at the expense of health to be satisfied,—all the powers of man's organization being brought into requisition and overtaxed in the daily struggle for self-maintenance and individual pre-eminence. In many instances where the labor is chiefly of a mental character, there is too often excessive strain upon the intellectual faculties; and should this happen in connection with hereditary predisposition to insanity, it is likely the mind may succumb, when its possessor will not only be thrown back among the crowded ranks of the unsuccessful, but may sink perhaps into hopeless lunacy. Indeed, in the professional man, whose bread is usually entirely dependent upon never-ceasing brain-work, the retrograde metamorphosis of tissue must be enormous; and should the slightest instability in his nervous system exist, the sum of his efforts may eventually cause a disturbance leading to the development of insanity. In this way may civilization be productive of the disease. It is estimated that, in civilized communities, the proportion afflicted is about one in five hundred.

4th. Sex undoubtedly exercises some effect; for instance, a nervous, hysterical woman is much more liable to become insane than a delicately-organized man exposed to the same influences. In debilitated women, especially in moments of joy or grief, you always have cause to dread incidental insanity. When a mother loses her darling child, the emotional disturbances are often terrible, and you may fear the result; but these apprehensions naturally do not extend to the father. Indeed, it is very improbable

that a man could be constituted with such a delicate, nervous, and excitable temperament as a woman: his very organization precludes this idea; and it is doubtful if he be capable of experiencing the same intensity and exaltation of the emotions as his more frail and tender partner.

5th. The *period of life*, also, seems to possess a certain degree of influence as a predisposing cause of insanity. At the time of puberty, and also among women at the appearance of their menopause, we often find the development of mental complications. In man, even, it is contended that an equivalent *change of life* occurs at a certain advanced age, though not always accompanied by a loss of sexual capacity, the sexual proclivities frequently seeming to be enhanced, as a prelude to approaching decay. At this time, in consequence of the activity of the retrograde processes, insanity seems markedly to increase.

6th. *Education* is the next cause to be considered. In deficiently-educated children (I refer principally to *moral* education), whose training has been neglected, whose propensities to evil have been gratified rather than checked, whose bad temper has been developed instead of restrained, and in whom any instability of the nervous element exists, the least exciting cause may bring about insanity. It is a mother's care and training which made us the men we are; and we can never sufficiently appreciate her tender solicitude, since but for her constant and untiring efforts we might have been perverse, if not insane. On the other hand, excessive mental strain, such as is sometimes required in the school education of our youth, may be equally pernicious.

7th. *Individual predisposition* is a cause to which I have already alluded. It is consequent upon hereditary tendency, or it may be acquired. Many authors state that at least fifty per cent. of the cases of insanity are of ancestral transmission; and I am inclined to the belief that even this estimate is underrated. It is also claimed that this inheritance is more apt to descend from the mother than from the father. Children are in much greater danger when their mother has been insane prior to their birth than when the disease appears at a subsequent period. Insanity is perhaps more readily transmitted to daughters than to sons.

8th. The *insane temperament* is that condition in which "an individual is, by reason of a bad descent, born with a predispo-

sition to insanity ; he has a native constitution of nervous element which, whatever name we give it, is unstable and defective, rendering him unequal to the severe stress of adverse events. In other words, the man has the insane temperament: he is liable to whimsical caprices of thought and feeling ; and although he may act calmly and rationally for the most part, yet now and then his unconscious nature, overpowering and surprising him, instigates eccentric or extravagant actions ; while an extraordinary and trying emergency may upset his stability entirely.”—*Maudsley*.

This physiological condition is most important in its medico-legal bearings ; and the defense of Joseph Fore—who was tried for murder in the first degree and acquitted—was conducted successfully upon this very theory. His subsequent actions and behavior clearly substantiated the fact that the plea was well grounded and justly maintained.

The *exciting causes* of insanity are divided into *physical* and *moral*.

1st. A very frequent physical cause of insanity is *masturbation*, a disgusting vice, to which, I must say, I believe an astonishing number of lunatics, either as a cause or as a consequence of their disease, are victims,—many more than the statistics of asylums show, owing probably to the false delicacy which causes patients and their friends to conceal the truth.

2d. *Drunkenness* is another of the causes of insanity. Men are often habitual drunkards before they realize it ; they then find it more difficult to dispense with liquor than with their meat and bread. Experience leads me to believe that inebriety as an exciting cause constitutes the principal factor in about one-third, if not in one-half, of the whole number of cases of insanity.

3d. *Epilepsy* is mentioned as a cause of insanity by some authors, while others contend that it is more frequently a result thereof. This we shall attempt to decide later.

4th. *Transmutation of nervous diseases*, so called by Trousseau, meaning that a nervous disease in one generation may be transmitted to the next or the second generation, but with a change in form. Hence we witness chorea in the children or grandchildren of epileptics, and *vice versa* ; these diseases not infrequently resulting in insanity. It sometimes happens that the transmission of

nervous disease may take a different form for each member of a family,—neuralgia in one, asthma in another, epilepsy or hysteria in a third, and in a fourth, insanity.

5th. That *chronic diseases* are remarkably productive causes of insanity is not an astonishing fact when you recall what I have stated as regards the necessity of a normal state of the blood in order to insure a healthy condition of the functions of the nervous system. Consequently, an anæmic or hyperæmic brain is incapacitated for a proper performance of ideation. The same result would happen in rheumatic or gouty affections, or in syphilis and other diseases due to a certain amount of blood-poisoning, whether the result of dyscrasiæ or of acute febrile diseases. Insanity, however, is by no means an inevitable result in all cases of this nature,—the absence or presence of the inherent predisposition playing the most important part in its superinduction. Then there is, as I before told you, a certain class of patients who do not remain permanently insane, but after an attack of insanity return to their normal condition, to relapse again, making it very difficult to anticipate with any certainty their future status.

6th. Derangement of the *sexual functions* has an extraordinary relation to insanity. In females especially many abnormal conditions of the uterus, such as retroversion, prolapsus, etc., are active agents in this respect. In curing the primary affection you will cause the entire disappearance of the insanity.

Van der Kolk relates a case of prolapsus, attended by melancholia, which was relieved upon the reduction of the prolapsus. But on removing the pessary the symptoms of melancholia immediately reappeared, to vanish again upon renewed reduction. This experiment admits of no doubt whatever, having been confirmed by the experience of numerous other alienists. There are women who become insane during every pregnancy: some before parturition, some immediately after, and others during lactation. These facts are of importance, and, whether he be a specialist or not, a physician cannot afford to ignore them, as their significance is evident.

7th. *Injuries to the head* are often the cause of the most insidious, dangerous, and intractable forms of insanity. I believe that all severe blows upon the head, sooner or later, may cause very serious brain-symptoms, though years may elapse before their appearance. In such cases, if you carefully analyze their histories,

it will be evident that the insanity was, beyond doubt, directly the result of the blow. Forbes Winslow contends that these disastrous and insidious consequences resulting from injuries of the head are too often overlooked.

The *moral cause* of insanity consists simply in too great violence of emotions, which, when occurring in persons possessing an inherent tendency to insanity, very seldom fails to result unfortunately. Among the more dangerous emotions we find those of anger, jealousy, hatred, and love very prominent. The sudden change in the tide of one's affairs, and the subsequent resulting sensations, have often caused reason to be dethroned. Not only the change from wealth to poverty, but also the reverse may result in mental disease. Indeed, the panic of 1873 has alone furnished me sufficient evidence of this fact. The excitements incident to war, and its disastrous consequences, have afforded ample evidences of insanity consequent upon undue violence of the emotions. In a word, whenever the mind of an individual is subjected to undue strain, or the passions are extraordinarily roused, other conditions being favorable, mental alienation may ensue.

LECTURE XI.

INSANITY—*continued.*

Two Primary Groups.—Ideational and Affective.—Affective Form the Foundation of the Others.—Change in Feelings characteristic.—Delusion no Criterion.—Hallucination.—Illusion.—Delusion.—Insanity should not be a Stigma.—Change in Normal Self.—Illustrations of the Affective Type.—Wrong Conception of the Insane.

GENTLEMEN,—In a former lecture I spoke of insanity as being capable of division into *two primary groups*, these groups being formed by variations of the disease, as affecting either the words or the acts of an individual. A person may be insane in his words, or may be insane in his actions; and this difference admits of the division of insanity into *ideational* and *affective*,—ideational insanity being evinced by irrational words, and the affective referring to actions, which are the manifestations or outcroppings of insane feelings. These facts we now propose to study; and they are very important. Indeed, if you have succeeded in seizing this distinction, you have a part of the subject of insanity pretty well fixed in your mind.

We have, then, ideational and affective insanity; the latter—also called the *pathetic*—pointing to impulses of perverted volition; the former evinced by irrational conversation or incoherent and erroneous reasoning. We might call this latter a disease of ideas (assuming the fact, however, that they are immaterial), but we have reference, of course, to insane words, in consequence of disease of certain cortical cells, made use of in the elaboration of thought. Hence the individual is ideationally insane, and can no longer think normally; this deficiency being manifested not in his actions but in his words. We find, moreover, that these groupings are not peculiar to any one of the forms of insanity, but to all of them, so that we may have either mania or melancholia of the ideational type, or of the affective type, or of both. I want you all to understand this fact, which is, according to the best authorities, that in any given case of insanity you may find it partaking

either of one or of both of these types. Melancholia may therefore exist, and only evince a disturbance of the feelings; but if it exist with delusions, there is then a complication of the ideational with the affective type, and mental action becomes perverted. On the other hand, a person may have mania or melancholia with delusions, and this will necessarily imply an erroneous ideation with aberration of sentiment, as expressed by both words and actions.

Dr. Maudsley teaches that in all cases of insanity we generally have first to study the affective form, as usually the underlying current. Persons, at the commencement, generally become insane through perversion and want of proper co-ordination of their feelings; and this condition almost always underlies all other forms of insanity. It is the deep morbid state, of which all the other phenomena are but the outcroppings. Hence ideational insanity is nearly always preceded by the affective form. Again, if the ideational form becomes developed, it is usually in combination with the affective form, and if it be cured by therapeutical or other agents the affective form may still linger, and be the last to disappear. Now, if this be so,—and we have much that is reasonable and probable to support it,—then the *affective form of insanity is the fundamental fact*, with which we must thoroughly familiarize ourselves to comprehend fully all the bearings of this subject. It lies at the bottom of all cases, constituting the root from which they spring, and in their analysis we must acquaint ourselves with its phenomena. The affective life will be the first to attract our notice, its derangement will complicate all other varieties of mental disease, and will be the last to remain disordered. When we seek to study a person, is it not always through his feelings we commence our observations? We analyze his peculiarities and idiosyncrasies, knowing that all his actions are the sum or product of these feelings, or rather spring from impulses controlled by them, which latter, by their continual indulgence, have the tendency to produce habits and customs often characteristic of the individual. You will now understand why emotional insanity is always manifested by perverted feelings and actions, and also will realize the importance which psychological physicians place upon the scrutiny of the first symptoms, which are the precursors of phenomena culminating in convulsive action and moral irrespon-

sibility. In fact, some affective form of insanity may be found at the bottom of or complicating every case we meet.

Some persons may be lunatics, yet not evince any ideational insanity; but a change in the feelings is always characteristic. If we take delusion as a criterion, we are liable to ignore some very dangerous forms of insanity, because there are many serious manifestations of the insane temperament, as well as certain affective forms of insanity, which will attract our attention only through actions following the irresistible impulse of distorted feelings. Delusions, therefore, cannot be correctly considered a criterion of insanity, since it may indubitably exist without their presence. This is a very important fact, with which you should be acquainted, because for a long time in England, as well as in America, delusion was always considered a *sine qua non* of insanity; and even to-day many prejudiced lawyers consider that its absence entirely invalidates the plea of insanity, and many very important judicial decisions, involving property, and even life, were erroneously based upon this principle. To-day, the old doctrine is rejected, almost entirely, by the best authorities, and the new one is receiving no little legal sanction; and if you adhere to obsolete ideas you will necessarily aid in impeding psychological progress. Having so often referred to delusions, it becomes necessary to define the word. The definition I will offer may not be precise or philosophical, nor exactly correspond with that of the books, but I purpose to present my own views, and in such a manner as to facilitate their comprehension and recollection.

There are *hallucinations* and *illusions* as well as *delusions*. An *hallucination* is the imagination of unnatural ideas, without any facts for their support, and totally opposed to reason. I might, for instance, suppose for a moment that innumerable rats were running over this table and that I could clearly see and distinguish them; but my reason would tell me that such a conclusion was without foundation, and therefore I would not believe it, as being opposed to common sense, but would reject it without hesitation. Now, in refusing the reality of such an idea I reject a hallucination which momentarily I actually experienced. An *illusion*, on the other hand, is a deception having a material or sensible basis upon which to rest, usually occurring through an imperfect or improper impression made on the senses; as when a person con-

ceives the notion that an odor, really present and penetrating, emanates from the infernal regions, or some "ubiquitous corpse." Those erroneous perceptions resulting in hallucinations and illusions, and resisting the investigation of the reasoning faculties, and, though opposed to common sense, persisting in spite of vigorous intellectual scrutiny and analysis, constitute *delusions*. Delusion is a strong presumptive proof of insanity, and is insanity in ninety-nine cases out of a hundred. If the delusion be persistent, the person will be chronically insane. There might be an exception to this law. "Delusion, exceptionally," says Winslow, "may be present and not be incompatible with sanity." A man might suppose that he was extremely handsome and exceedingly intelligent, and still be homely and stupid. Such a misconception might not necessarily be considered insanity. Yet such an exception does not invalidate the rule already established in reference to the significance of delusions. They constitute the only cognizable feature of insanity, according to old English laws; but in the more enlightened jurisprudence of our day we do not attach to their absence the same importance. Some of the most terrible and dangerous forms of insanity undoubtedly exist without any delusions, and are only manifested pathetically, yet prevent the individual from remaining in harmony with his social sphere, render him dangerous, and necessitate his isolation, to preclude the possibility of his being aggressive and dangerous to others. Of course, insanity should not involve the least stigma upon a person. It is purely a misfortune, as much so as smallpox or any other disease (perhaps it is less of a calamity than syphilis), being an abnormal physical condition of the brain, with a co-existing instability of the nervous system. In contradiction to those old theories, but in accordance with modern enlightenment upon the subject, I tell you that insanity *exists only* when a person is *changed from his normal self*.

We have now conclusively shown that a man may be ideationally insane and still possess no marked delusion. Without delusion he may nevertheless be a hopeless and dangerous lunatic, and the fact may become patent by the perpetration of some terrible crime, for which he could not be held morally or legally responsible: his powers of volition are deposed, and he acts under the influence of diseased cortical cells. But in such a case there has

existed, as a *fundamental fact*, a change in the individual's normal self; and although no apparent impairment of intellect was manifest, and the reasoning faculties *seemingly* remained perfect, this alteration, though unnoticed by anybody not especially interested in and acquainted with the individual, will have been very evident to the intimate friends and relations from the very inception of the malady. You might, without recognizing his insanity, hear a man addressing a jury very eloquently, lecturing on scientific subjects, or preaching the gospel to an intelligent audience, or, while engaging in the gayety and pleasures of the ball-room, by his wit and conversation attracting the attention of all bystanders, and yet all the time he might be laboring under some serious form of mental aberration. In such an insane person there is little ideational insanity, but there is a morbid change of feeling and sentiment, which might make him dangerous, and even induce him ruthlessly to take human life. Of course there has been a change from his normal peculiarities and type; and though the fact may not be known to the multitude unacquainted with his habits, still, his intimate relations realize that he acts rather strangely and inconsistently, and that he needs watching. That such cases happen is undoubtedly true. What I am trying to make you understand is, that this change in an individual's normal self is of great importance, pointing, as it does, directly to insanity. Of course, you cannot judge of this unless you are familiar with a man's habits and antecedents; but you should in all cases become conversant with his usual deportment, his normal social, moral, and intellectual states, and the history of his former life, before you feel justified in arriving at any definite or safe opinion. All these facts are of great weight, and it behooves you to have positive knowledge of them before framing any conclusions. At the present day physicians are often called upon to testify, and medical knowledge is frequently required in evidence before courts of law: hence you cannot afford to be tossed about by every wind of doctrine.

Recollect, therefore, that perversion of sentiments or habits, or perpetration of acts socially absurd, immodest, or indecent, points to insanity in some persons, while it does not in others. Suppose, as Blandford remarks, that an Italian brigand cuts a man's throat without the least regret, it does not prove insanity, though the act

is brutal and characterized by absence of emotion, and is not followed by qualms of conscience, as also might or might not be the case in insanity. Why is this not insanity? Because there is no departure from the man's normal self. On the contrary, murder is his habitual occupation; and by his often-repeated crimes his conscience has become blunted and hardened, so that he murders without compunction. But if some eminent and learned divine, distinguished for his social virtues, were, without motive, to cut a person's throat, we would conclude that he was either a consummate hypocrite or a confirmed lunatic, and the study of his life would inevitably confirm one or the other hypothesis. Or suppose a parsimonious man, taking care of every dime, possessing a ceaseless solicitude for his worldly goods, never giving or expending anything except for the absolute necessities of life, were suddenly, without adequate motive, to become excessively liberal, squandering his money as recklessly as formerly he treasured it carefully: would not this point to a suspicious perversion of feeling and to a change of sentiment consistent only with insanity? Hence the significance of actions, especially when contrasted with the previous conditions of a patient.

The foregoing illustrations point especially to the development of insanity of the affective type, without any particular concomitant evidences of the existence of morbid ideation, which is expected by a great many, especially inexperienced students, to constitute an inseparable part of the picture of insanity. Many imagine that in order to be insane a man must be a raving maniac, loaded with chains, incoherent in speech, and wild in demeanor. Such persons have the conception of only a single form of insanity,—that of acute mania. But in the affective variety there is not necessarily any raving, or any other extraordinary behavior. I hope you all now understand the distinction between *affective* or *emotional* and *intellectual* or *ideational* insanity: the last pointing to disease of those portions of the brain which preside over the intellect, the former to disease of those parts which elaborate the feelings or emotions.

There are different opinions, all of them strictly conjectural, in regard to the location of these different faculties. It has, for instance, been asserted by some authors that the seat of the moral faculties is in the medulla oblongata. Maudsley says, "Certainly

there do not appear to be satisfactory grounds, either in psychology or physiology, for supposing the nervous centres of emotion to be distinct from those of idea." These are nothing more nor less than hypotheses; but wherever these faculties do reside, whenever their seat is diseased the pathetic form of insanity is developed. We have said that a man may be morally, and yet in no respect ideationally, insane. Whether or not we can make this arbitrary division is a question still *sub judice*, many writers taking the ground that when one portion of the brain is diseased the morbid process probably extends to and involves all the contiguous parts. This is not necessarily so, though we can hardly conceive of one part of the mind being affected without at least an implication of its entirety. It is therefore believed by some that affective insanity is always complicated by some defective ideation, which may, and probably does, exist without being very apparent.

Before dismissing the subject, I will advert to the fact that in insanity the feelings are always first affected, and that it is through them we must study all collateral phenomena. When an insane person commits homicide, suicide, rape, or arson, it is the result of some serious form of insanity; but, nevertheless, it is only the outcropping or symptom of a morbid condition of the moral nature, and delusions are therefore not the only evidence, but are simply symptomatic of ideational insanity. Overt acts of violence indicate a morbid condition of feeling,—incoherent words, a morbid condition of thought. Or, to make it plainer, just as *insane words* are produced by *insane thoughts*, so are *insane acts* produced by *insane feelings*.

LECTURE XII..

INSANITY—*continued*.—MELANCHOLIA.

Phenomena.—"Concrete Form of Misery."—Difference between Melancholia and Mania.—Lypemania and Pantophobia.—Hypochondriacal Melancholia, Phenomena of: Case of.—Another Form of Melancholia.—Tendency to Suicide.—Melancholia Attonita.—*Folie Circulaire*.—Paroxysmal Violence not necessarily an Evidence of Insanity.—Affective Form never absent.—Moral Treatment: Evil Effects of Delay, Beneficial Effects of Asylum Treatment.—Medical Treatment: Opium, Aloetic Laxative, Alcoholic Stimulants, Chloral for Insomnia, Rhamnus frangula.

GENTLEMEN,—To-night we will commence the consideration of the different forms of insanity. The first one I shall speak of, and one with which you will frequently meet, is melancholia. Melancholia, as the name denotes, is characterized by great mental depression, excessive grief, and painful sadness. It belongs, for the most part, to the *affective* or *pathetic* variety of insanity, as it relates more to the feelings of the individual than to his intellect.

The first phenomena to be observed are, peculiar mental depression, vague and indefinable oppression, strange and indescribable sorrow. There is a condition of lypothymia which the patient cannot shake off, "an oppression of the self," and his despondency is to himself something altogether unaccountable; he is at a loss to state why he is so sad, or why he experiences this vague and indefinite gloom; in short, he is entirely overwhelmed and completely overburdened, life becomes a misery, and existence is unendurable. This general affective dejection may constitute the entire morbid state, no delusions existing, the feelings only being perverted without any implication of the intellect. Delusions produce a form of insanity in which we always presuppose an affection of the intellect; that is ideational insanity. As Maudsley says, after the mental depression of melancholia has existed for some time, "*a concrete*" form of misery is produced; the patient seems to assume some fixed and settled idea, or develops a terrible, all-absorbing delusion. This concretion is indicated

by delusion, and suggesting, as it does, a hypothetical cause for the overpowering anxiety of the mind, far from increasing the grief or depression, seems generally to afford relief. This delusion, however, bears no relation to the intensity of the melancholia. In some persons it appears as a very trivial matter, in others it is "expressive of some great fear or suffering." We find some melancholic patients laboring under the belief that they have committed some grave offense, or that they are guilty of "the unpardonable sin." They imagine that they are suffering the torments of the damned, and experience these tortures for some trivial delinquency, which ideas are absurd of course, but greatly exaggerate their distress. In melancholia, which purely appertains to the affective variety of insanity, the morbid manifestations are mainly evinced in the actions of the patient, while in mania, the intellect being affected, the disturbances are chiefly exhibited in the words, as expressive of thoughts.

Melancholia, as met in general practice, at the bedside, or in the hospitals, is capable of being separated into two original groups,—*lypemia* and *pantophobia*. *Lypemia* consists of melancholia always attended by delusions. *Pantophobia* refers to a condition of the patient which is characterized by a constant dread and fear of everything, without any definite cause for apprehension. The latter is melancholia without "concrete form." Remember that this division is made by Maudsley simply for practical purposes. I have already told you that there are two grand primary divisions of insanity,—the affective form, relating to morbid feelings, and the ideational, relating to perverted intellection. All cerebral disorders resulting in insanity come under the one or the other, or under both, of these forms. This being the case, it follows that just as we have melancholia without intellectual insanity, so do we have mania sometimes with only the signs of disordered actions, and purely pathetic.

However, when mania exists, it is almost invariably in the form of intellectual insanity; still, the precursory manifestations are emotional, and we have acts expressive of deranged sentiments, affections, habits, etc., prior to other developments. In mania, therefore, when affective in character, the perverted feelings are preternaturally excited; in contradistinction to melancholia, in which they are depressed. Therefore, when the insane feelings

are *excited*, there is an affective *mania*; when they are depressed, there is an affective *melancholia*. If now in affective melancholia delusions supervene, the intellectual powers becoming impaired, the faculty of reasoning is involved, the conversation is more or less irrational, and then intellectual insanity has appeared and complicated the case.

Several times have I already stated, at the risk of being monotonous, because I wished you all to recollect it, that Maudsley subdivides insanity into ideational and affective. In the first case there is perversion of intellect, and in the second, perversion of the emotions or feelings. Now, the first phenomena of melancholia refer to disturbed feelings, through grief, sadness, despondency, etc., without any intellectual derangements. Of course, the patient is not, as yet, ideationally insane; but delusions may sooner or later complicate the case, and we then have ideational insanity, and the patient's abnormal condition will be manifested alike in words and actions.

Another subdivision of melancholia is the *hypochondriacal*. In this form we find the individual studying, criticising, examining, and investigating his own real or supposed bodily ailments. He imagines himself the victim of some disease; perhaps he has a "fluttering of the heart, a film before his eyes," etc., and all sorts of indescribable troubles annoy him. He watches these hypothetical symptoms and interprets them in his own way, and, when consulting a physician, will often give explanations of his disease which are really remarkable. It occurs in the vast majority of instances that a melancholic patient may be only slightly hypochondriacal, and yet be entirely unable to attend to his business or pursue the ordinary avocations of life. Of course, this constant, self-absorbing preoccupation becomes a source of disease, and results disastrously, by inducing certain changes in one or more organs. The incessant concentration of the mind on the feelings develops delusions, and, acting under the influence of such perverted emotions, the patient may be led to the commission of rash actions with terrible consequences. Whence do our actions spring? Whence our impulses? They originate from the feelings; hence the perverted feelings, stimulated by a morbid influence, will sometimes induce a patient to commit suddenly some terrible crime, and this may occasionally happen even in the

slightest form of hypochondriasis. It has occurred, as related by Maudsley, that a patient cut open his abdomen with a piece of glass, penetrating the intestine, simply "to let out the gas."

Now, when the feelings are thus perverted so as to constitute disease, whether slight or intense, it gives the physician great annoyance; and many hypochondriacs out of asylums will come and torture you by a detailed account of their troubles. The face will be pinched and anxious, the story related you will have perhaps heard a hundred times reiterated, and know it by heart, and yet you must patiently listen. To treat such people is, indeed, a task, though it is at times exceedingly interesting to observe their peculiarities. A short time ago I had under treatment a hypochondriacal gentleman, who stated that he was suffering from muscular rheumatism to such an extent that all locomotion was impeded. This pain had resisted the iodide of potassium, and all other treatment. The other evening I saw him; he recollected that he wanted to go to a certain place of amusement that very night, which inclination so absorbed his thoughts that he overcame his morbid feelings, jumped up suddenly, walked off briskly, and was cured, until, his interest being no longer excited by extraneous matters, the old trouble returned, because his thoughts reverted to the same morbid channels. All cases of insanity are not sent to the asylum for treatment. Some wealthy families will often seriously object to, and strenuously oppose, a removal of one of their members; their station in life enabling them to furnish all possible conveniences and comforts to the patient.

There is another form of melancholia, which you may meet with in the asylums as well as in private practice, and which you should be able to recognize with facility. I allude to *melancholia with excitement*, which is sometimes not readily distinguished from mania. Delusions will appear, and the patient become very prone to violence; a dangerous homicidal propensity will be developed, and intense mental anguish may culminate in suicide. This morbid tendency to the consummation of some terrible act is engendered by the desire to relieve the crushing depression. I remember a case in an asylum, where such a patient, previously considered harmless, actually knocked a man down and beat his brains out with a chair before assistance could be obtained. When asked by the coroner his reason for committing this crime,

and if he had any spite against his victim, or had any previous quarrel with him, he simply answered, no: that, on the contrary, they had always been good friends, and it was only the pent-up feeling, the excruciating mental torture, which he by some act desired to relieve, that caused him to commit this deed of blood. Some insane people will perpetrate a murder in order to become victims of the law, perhaps with the hope of being hanged, to rid themselves of their misery. It has happened that mothers have destroyed their young children simply to send them to heaven, or to relieve them of the pangs of hunger or poverty. Having thus seen the consequences of perverted emotions, you will readily understand why the affective form of insanity is so much to be dreaded.

Another danger in melancholia, and one against which you must always be upon your guard, is a tendency to suicide. This impulse has to be thwarted; and the actions of all melancholic patients are to be closely watched on this account, as the intensity of their mental suffering is sometimes so great that they frequently attempt, and but too often succeed in accomplishing, their own destruction. They resort to cunning artifices of every description for the accomplishment of their purpose, picking up pins and needles from the floor, and concealing all sorts of objects, with which at night they will attempt to destroy themselves. I have known them to secrete a table-knife, to use at the first opportunity when unobserved; and in my own experience I recollect a patient who cut his throat from ear to ear with a knife which he had slyly abstracted from the dinner-table and previously sharpened on the window-sill. Indeed, the more closely they are watched, especially if they suspect it, the more persistent are they in their efforts at concealment and deception. Hence a sleepless vigilance alone will be successful in frustrating their destructive propensities.

Another form of melancholia, and one which might be confounded with dementia, is *melancholia attonita*, or melancholia with stupor. Life in such patients seems to be purely vegetative; they will take no food, refusing even to swallow it when it is placed in their mouths. At times they will resist all efforts to introduce aliment into their stomachs, from fear of being poisoned, imagining that some relative or friend wishes to kill them. The

peculiarity of this variety is that the patient is in a seemingly cataleptic condition,—a *quasi* stupor or somnolent inertia,—and on account of his immobility forcibly reminds you of a statue. Once having assumed a position, the patient will maintain it for hours, perhaps for a whole day, or until utterly exhausted. There is a striking inanimation, the organic functions appear almost to be held in abeyance, the animal actions are suspended, and the face is a blank, without the least expression or play of emotion. There is no consciousness of time, locality, or personal identity; the patient has not the least idea of his surroundings, sometimes involuntarily swallowing anything placed upon his tongue, and at other times it will be impossible to make him eat, until you at last resort to the stomach-pump, introducing food mechanically. At times the patient will temporarily revive, be like a person awakening from a sound and protracted slumber, during which he has apparently been dreaming, and will make inquiries which undoubtedly point to his previous unconsciousness. His mind has been engrossed in one terrible delusion, which wholly absorbed his faculties; but one feeling, one idea, has prevailed, in which his whole being has been concentrated, and since, as Dr. Maudsley remarked, to be wrapt up in one sensation would be equivalent to the possession of none, we can understand the profundity of the patient's lethargy. This form of melancholia is really painful to witness, and the immobile and cadaverous appearance of its victims is well calculated to arouse sympathy.

Still another form of insanity to be considered is what is termed by the French *folie circulaire*. In this form there is a certain alternation in the manifestations from melancholia to mania, and it may be a difficult matter to differentiate between the two diseases. At one time there may be an exacerbation characteristic of mania, followed by a period of mental depression. I have witnessed the interchange in this type of insanity develop itself towards the termination of protracted cases of melancholia, and in a case of five years' duration of the latter affection, now convalescent, *folie circulaire* was the immediate precursor of the present favorable condition. The case to which I allude was that of a distinguished divine, and was the most profound I have ever witnessed, being for eighteen months, in the earlier periods, *melancholia attonita*. *Folie circulaire*, however, rarely follows this

satisfactory course, as in many cases of inveterate and incurable insanity it will be present.

At this juncture, perhaps it would be well for me to caution you against supposing that acts of paroxysmal violence necessarily constitute a form of insanity. This would be an erroneous conclusion, as these explosions are purely symptomatic, really forming but a single feature of the malady, and in some cases are entirely absent: just as in ideational insanity we do not necessarily find delusions, though they are generally present, so in some cases of affective disorder murderous and suicidal and other morbid propensities may not be at all manifested. The old subdivision of certain forms of insanity into pyromania, erotomania, suicidal and homicidal mania, etc., was indeed unfortunate, and calculated to lead to erroneous conclusions: we now know that the propensities constituting those states are only symptoms common to many, and hence not in the least peculiar to any special form of insanity. Just as I told you in previous lectures, that the most dangerous forms of insanity may and often do exist without delusion, so are these last-mentioned conditions often entirely absent. Madmen are often able to *regulate their conversation* perfectly, speak coherently, and often astonish you with the brilliancy of their thoughts, but they cannot *control their actions*; and hence the affective form is never absent, but pervades all forms and features of insanity.

To resume: in insanity the affective type is almost always at the bottom of the difficulty, developing perverted emotions and change of habits. This state may be very dangerous without any evidence of intellectual disorder. In short, it complicates all the varieties of insanity, preceding, accompanying, or following them; thereby greatly influencing their course and deeply impressing with its peculiar influences their manifestations. These are very important facts, and when you understand them a great part of the subject of insanity is clear to your minds.

A very important fact to remember is, that the moral treatment constitutes the most important feature among the influences that are required for the amelioration of this form of insanity. The moral treatment is essential, as the perversion in sentiment is the fundamental condition of the disease. In what does this treatment consist? In isolation, seclusion, firmness combined with

kindness, and discipline of mind and body. I have already told you that when a person is insane he is no longer in harmony with his social relations; there is something out of gear, and he may become a source of danger to himself and others. He should, therefore, be isolated, and treated by those who understand his affection. The loose screw should be accurately adjusted, and the machinery of the brain be oiled and reconstructed, that future harmony may prevail. As general practitioners it is your first duty to know this, and I cannot too persistently dwell upon it. Many an unfortunate being, through neglect, injustice, or ignorance, has been doomed to the miseries of chronic insanity; compared to which all other sufferings are small, and the agony of death itself infinitely preferable. Yes, death is a thousand times more desirable than a life embittered by chronic insanity and rendered desperate and horrible by the perpetuity of its wretchedness. If this were the only benefit you have derived from these lectures, you would have accomplished much, and would have acquired a knowledge which, though apparently of little value, is very important in its bearings from a humane point of view. When the patient, consequently, is in the inceptive stages and the disease is still acute, he should be immediately sent to an asylum, in order to prevent the occurrence of hopeless mischief. Why is it that the number of inmates of county and State asylums is constantly swelling? Is it not due to the neglect in this regard of physicians, or to unfortunate legislation and the endless amount of "red tape," by which the destitute are left without proper treatment until the disease is no longer acute, but chronic,—its difficulty to control being in exact relation with its duration? By this time the patient has become not only a permanent burden to his family but also a life-long curse to himself, and the asylum has finally to open its doors for him to enter, perhaps never again to leave. Treat early, therefore, remembering that when three months have passed insanity ceases to be acute, and that success depends upon immediate and energetic treatment. I may here state my thorough conviction that some cases of melancholia may recover without being sent to an asylum; but this is the exception, and does not invalidate the rule. For, while it is true that a rich man may have a comfortable residence, faithful attendants, and eminent physicians to treat him, how many can afford these condi-

tions of home treatment? and is not the number of the suffering and poor infinitely greater? Moreover, even under the best outside treatment, from the inexperience of nurses, suicide will but too often result.

There is often a prejudice against sending persons to lunatic asylums, from the fear that their sudden removal to such associations may prove pernicious. On the contrary, the moral effect is oftentimes excellent: the patient recoils; there is a beneficial shock, which occasions him to enter into himself and reflect, "Why am I here, in the company of persons so plainly mad?" It causes an introspection, having a most salutary effect, to which the order and discipline of the place are also auxiliary. Then the moral measures are conducive towards teaching the patient to exercise a certain amount of self-control and make efforts to recover. You may exhaust the therapeutic resources of the pharmacopœia, and, if the patient do not try, he will not improve; and it is this endeavor towards restoration for which I look as the first auspicious sign of dawning reason. If the patient evince no energy, he will not get well; but if he try to disperse the mental mist which surrounds him, he will progress favorably, and his endeavors will to a great extent be advanced by the moral treatment he receives.

The insane should be treated like children, with kindness and firmness, with uniform affection and courtesy; but the necessary exactions must be unswervingly enforced, and, like children, they should be rewarded for their good behavior and rebuked for their disobedience. They often will do better, or at least, like children, will try to do better. When depressed, you should encourage them and relieve their mental anxiety, and when unruly, you must check them, but without undue severity. This is far more important than the *medical treatment*, in regard to which I must say that I have not unbounded faith. I have tried opium, the so-called sheet-anchor in melancholia, and all its vaunted preparations, but with no brilliant results. One thing is certain, as maintained by Schroeder van der Kolk, that the disease is often due to a loaded colon, accompanied by obstinate constipation. In such cases an aloetic laxative, or *Rhamnus frangula*, the laxative *par excellence*, will generally relieve the constipation and cause mental cheerfulness to reappear.

As in the affective variety of insanity there is great mental sorrow and physical prostration, alcoholic stimulants can be beneficially administered. Melancholic patients bear them remarkably well, and to an extent truly astonishing. According to Blandford, such patients should receive alcoholic stimulants at every meal,—either rum, brandy, or whisky. Of course, there is a medium in everything, and so in the administration of these stimulants you should bear in mind that what may be too little for one may be too much for another. I have always found the breath the best guide, in this as well as in acute and febrile diseases, to determine the proper regulation of the remedy: if surcharged with alcoholic fumes, it is not being appropriated, the system is surfeited, and its supply should be correspondingly curtailed. Conquer the insomnia at night by the judicious administration of chloral, remove all causes of the disease which you can ascertain, and by a liberal administration of nitrogenized food, especially in the liquid and concentrated form, seek to maintain the vital forces in the best condition possible to resist the inroads of the malady.

LECTURE XIII.

INSANITY — *continued*. — MANIA. — MONOMANIA. — DEMENTIA. — MORAL INSANITY. — IDIOCY. — IMBECILITY.

Acute Delirious Mania. — Treatment. — Mania: its Characteristics, Course, Prognosis, and Treatment. — Monomania. — Dementia, Acute and Chronic. — Moral Insanity: Diagnosis of; Illustrative Case. — Idiocy. — Moral Imbecility.

GENTLEMEN, — In my last lecture I spoke to you about melancholia; to-night I begin with the discussion of a different variety of insanity, known as *mania*. For practical purposes we divide mania into *acute delirious mania*, *acute mania*, and *monomania*.

Acute delirious mania is that form in which maniacal symptoms exist with more or less elevation of temperature; in other words, when superadded to the ordinary symptoms of mania there is more or less heat, which can readily be detected by the thermometer. This acute delirious mania might more properly be called typhomania, on account of the invariable existence of adynamic symptoms, very often proving fatal. The symptoms are typhoid in character: the pulse is rapid and frequent, the tongue dry, and asthenia becomes more or less prominent. It is well to understand this condition, as it requires immediate and appropriate attention. Indeed, the disease often runs such a rapid course that there may not be sufficient time to effect the removal of the patient to an asylum. It is a matter of life and death, and, unless earnest measures are resorted to, the patient rapidly grows worse and sinks.

In acute delirious mania there is an active delirium accompanying the symptoms of mania not unlike what we meet with in acute febrile diseases. The delirium which is such a prominent symptom in certain low forms of fever should not be mistaken for that of the disease under consideration, as the manifestations of the latter are much more active, and an absence of enteric complications constitutes a marked feature. Although the thermometer may run up to 105° F., the pulse be frequent and rapid, and

sordes accumulate upon the teeth, by being on your guard you can nearly always make a correct diagnosis.

The disease usually is initiated violently and suddenly, and runs its course in a short time, sometimes lasting three or four days, at other times a week or longer. The aspect of the patient, history of the case, and peculiarities of the delirium will enable you to differentiate this affection from delirium tremens, meningitis, or ordinary cases of mania. The presence of tremor, peculiar visual hallucinations, and good-natured delirium in the first, the different character of the symptoms in the second, and the absence of fever and asthenic symptoms in the third, will enable you to avoid errors of diagnosis.

We should freely use stimulants, give nutritious, supporting food, and by all means produce sleep, which is always absent, and very difficult to secure. Obstinate insomnia is found in all forms of mania, and is frequently a pertinacious symptom. Of all the therapeutic measures used to promote sleep under such circumstances, I am convinced that the most efficient is the *hydrate of chloral*. Of course, it should be administered cautiously, and not be given in such large doses as were recommended by Sir James Y. Simpson; for though chloral is an invaluable addition to the pharmacopœia, it is as potent for evil as for good, and care as well as judgment should be observed in its use.

It should not be forgotten that while we have to keep up the patient's strength by good and nutritious food, the diet should be one easily digested and assimilated, and that concentrated liquid aliment, such as beef-tea, milk, alcohol, etc., is indispensable.

Oftentimes persons afflicted with acute delirious mania become so violent that one is at a loss to know what to do with them. One of the best measures to be resorted to is that recommended by Blandford and Shephard,—the wet pack. Dip a large sheet in cold water, and completely envelop the patient in it. After it has been on for an hour or an hour and a half, the patient being wrapped in numerous heavy blankets, so as to induce free perspiration, the temperature will be reduced and the active delirium relieved. Of course, care must be taken that the water be not too cold, that the application be not continued too long, and that the patient be not exposed to taking cold afterwards.

Ordinary mania, such as you will generally meet with in asy-

lums, as well as in private practice, may sometimes resemble acute delirious mania; but in the former there is no fever, neither is there a rapid pulse or a heated skin. Ideational insanity, generally preceded by affective insanity, exists in both.

Now, what are the *characteristics of mania* which may enable us to discern it without difficulty? All cases of mania present more or less excitement,—mental excitement,—evinced by incoherent conversation, showing an irrational state of the mental faculties and a morbid association of ideas, to which are added wild gesticulations and an almost constant state of motion. Maniacs dance, sing, and jump without adequate cause; their actions are violent and excited, their fury being quite beyond their control. These are the usual symptoms, the ideational insanity being plainly shown by their conversation; but there is, nevertheless, a perversion of the affective life underlying the disease and deeply impressing all its manifestations. This necessarily displays itself in actions, and the patients are restless and destructive. They are difficult to manage, and their volubility is truly astonishing. This fact caused the older writers to contend that in maniacs the memory, as well as all the other mental faculties, became enhanced or brightened. Although such people show a remarkable mental activity, speaking not infrequently in rhyme with astonishing facility, still, this is no proof of the assertion, but simply points to abnormal increase of functional activity, resulting from disease. The mental machinery is out of order. There is a deficiency in the intellectual co-ordination, due to an absence of its controlling power. Not only is the presence of co-ordination necessary to the regularity and precision of muscular action, but the mental actions must also be under the same regulating influence, and in the cerebral hemispheres such centres undoubtedly exist. When they fail to act, or act imperfectly, there is, figuratively speaking, a screw loose in the brain, or a lever out of order; and, though the machine may be in rapid and continuous motion, with a striking evolution of thought and brilliant scintillations of fancy, still, the ideational workings are abnormally performed, and the intellect is impaired. We have here a condition of irritability of the brain, the same as we found in hyperæmia, or in inflammatory conditions of its membranes. You remember that when speaking of them I told you that the symptoms of irritation pre-

ceded those of depression. So we have in mania a certain hyperæsthetic state of the nerves of special sense, and of the perceptive and psychical powers, giving rise to the symptoms of irritation, whose pathology is very similar to that of congestive diseases. There is, of course, an increased flow of ideas, the mental powers are acting abnormally, and there is a greater scope, though less perfection, of activity, which corresponds with the concomitant symptoms of irritation. But, as its course proceeds, the patient is finally completely lost in the labyrinth of mental disease, and dementia appears, which state corresponds with the symptoms of depression, and fatuity may be anticipated. In maniacal excitement, and the dementia which follows, there is first an increase, and then a decrease, in the mental activity, invariably accompanied by a very imperfect performance of the intellectual and moral functions.

To illustrate, suppose the case of a man who in rowing a boat, instead of making slow and regular strokes with the oars, lacks the necessary co-ordinating power of muscular action, and makes violent and spasmodic efforts, which necessitate a considerable expenditure of strength, with little or no progress; several of his strokes would hardly equal one regular and natural sweep of the oar. In mania there may be more mental activity, but its astuteness, far from being increased, is diminished.

The most prominent symptom of mania is the mental excitement, and, corresponding with this, we always find an exaggerated feeling of self-esteem and self-exaltation. The patient, in his own estimation, is better than anybody else; and there always exists a presumptive superiority of some kind. Schroeder van der Kolk claims this as an indispensable symptom. Obstinate insomnia, especially in the earlier stages, is rarely absent. With this fact all physicians treating insanity are familiar. Troublesome constipation is usual. A remarkable tendency to the manifestation of thoughts by actions is characteristic of mania. Certain actions, representing thoughts, almost simultaneously accompany their utterance in words. Such patients therefore gesticulate violently, and act while speaking. The impulse to incessant motion is truly remarkable, and sometimes quite dangerous, which fact is interesting in a medico-legal point of view. "Thought is father to the impulse;" but here the thought is hardly recognized before the

muscular action gives it expression. It is in consequence of this that these patients run, dance, sing, kill, and burn. Because of this tendency to the exhibition of thought in external action it is that they are in constant mischief and trouble. I hope you will all seize the important distinction, that when the violence of action manifests itself by suicide, homicide, etc., such is not the only expression of the insanity; other well-marked symptoms will be found to co-exist. Dangerous propensities alone do not constitute the necessary evidence of the existence of insanity. On these grounds I exclude pyromania, erotomania, dipsomania, homicidal and suicidal mania, as forms of insanity, simply because they embrace only a single sign or symptomatic indication of the existence of unmistakable insanity; just as cough does not constitute pneumonia or bronchitis, but is only one of its accompaniments.

The course of mania is very uncertain, as it may last a week, a month, a year, or even a lifetime. The tendency of the disease is either to a favorable termination or to chronicity; or it may result in dementia,—the “tomb of reason:” once herein engulfed, man is no longer a rational creature, and that divine attribute which once distinguished him from the animal is lost forever.

Of course it behooves a practitioner to treat the disease in the early or inceptive stage whenever possible. The moral treatment is as indispensable in mania as it is in melancholia, and hence asylum discipline should early be resorted to. Mania, to some extent at least, seems to yield to therapeutic measures, and the bromides of potassium, sodium, and lithium have been employed with advantage, on account of their sedative powers, and their controlling influence over the circulation of blood in the brain. When we come to study the pathology of insanity, we shall see that there may be not only too much blood in the brain, but also too little, or that the blood may be poisoned, or abnormal in quality as well as in quantity. It follows that while the bromide of potassium may in some cases be serviceable, in others it will be productive of harm; as, for instance, when an anæmic condition of the brain exists.

The insomnia must be combated; and I have already recommended hydrate of chloral as the hypnotic *par excellence*. Free pustulation of the head, by the application of croton oil to the scalp, often acts marvelously in relieving the patient, especially

when the disease is becoming subacute in character. Sulphate of copper, ergot, and Indian hemp, combined with the bromides, as recommended by a distinguished foreign authority, cold affusion and tepid baths, cautiously administered, the hypodermic injection of morphia, and hyoseyanus (oftentimes exceedingly efficient), are remedies which have all proven useful in my hands whilst treating intense maniacal excitement. When the motor centres are seriously implicated, I believe that conium will produce striking results in quieting the restlessness, jactitation, and general excitation. As tonics in subsequent stages, I have found none to equal cod-liver oil, dilute phosphoric acid, quinine, and the preparations of iron.

The hygienic condition should not be neglected. Never forget that the excitement is accompanied by excessive retrograde metamorphosis of tissue, and keeps pace *pari passu* with these destructive changes. In acute mania, therefore, you should always administer large quantities of nutriment; and, where the patient resists it, it is your duty to compel him to take it, even if you must have recourse to the stomach-pump. Often sudden death occurs from intense excitement, when followed by corresponding depression and exhaustion. Whenever very intense activity exists, whether functional or organic, you have reason to dread the consequent reaction.

The prognosis depends greatly upon the duration of the disease, the age of the patient, and the number of previous attacks. If the family history point to insanity, you very reasonably will fear a relapse, and hence will give a guarded opinion; one attack of insanity always predisposes to another, and this more particularly when the patient is of an insane temperament.

MONOMANIA.

What is monomania? It is a partial, a delusional form of insanity; and the name is derived from the Greek words *μονος* (single), and *μανια* (mania), as it was supposed to be an insanity upon one particular point, or a disease of only one portion of the brain. There exists in monomania an apparently clear intellect, and even an unusual mental activity is oftentimes enjoyed, except upon a single point, or perhaps a few points, indicative of the patient's insanity. This form has also been called *delusional*

insanity, because the disease is always expressed by one or more delusions. Overweening self-esteem exists in monomania even to a greater degree than in mania. It is an almost invariable concomitant of the disease, and I have never seen a case in which it did not constitute a well-marked feature. Such persons grow exceedingly angry if you differ with them in opinion. They are often very intelligent, and may astound you by the acuteness of their reasoning powers, their general information, and their brilliancy of thought.

Are we to understand that there can really be a partial insanity? Can an individual be *non compos mentis* to a limited degree and mentally sound otherwise? Can there exist an absolutely defined partial pathological condition of the mind? Can a man be insane in one or two ideas and mentally sound in all others? I am not inclined to believe such a doctrine, but rather think that in this affection the mind is almost in its entirety impressed or biased by the delusion or group of delusions. It does not follow that because a man is insane seemingly upon but one subject he is sane in all other respects, since we cannot measure the extent of his irrationality, or positively assert that so much does or does not exist. We cannot restrict the limits of a diseased mind by any arbitrary barrier. I cannot conceive of a person's laboring under a delusion without there being a greater or less implication thereby of some other, if not all, of his mental faculties. This may not be apparent on examination, but, for all practical purposes, we can accept it without hesitation.

By way of illustration, I may remark that I knew a man who was on trial for some crime he had committed: he was examined by a committee of experts, who, after a careful investigation, concluded that he exhibited no evidences of insanity. A relation of the accused, however, suggested to his attorney to ask him "why he was more powerful than any other man." His manner immediately changed; and he replied, with great vehemence, that he held his power from the source of all glory, and was on terms of social equality with the Holy Ghost, and with other members of the Trinity, being in the habit of treating them to the best of liquors! Men will labor under such delusions, believing them as firmly as you would a mathematical fact. All the regions of the mind are, to some extent, pervaded by such delusions, when they

exist; and a man in such condition must, in some degree, be mentally unsound in every other respect. A person laboring under monomania, though apparently sane in many particulars, is, nevertheless, entirely *non compos mentis*, and ought, in most instances, to be secluded in a lunatic asylum; for when subjected to the soothing influences of this retreat he may cause little or no trouble, but if brought in contact with the world, where he would be liable to be crossed, he may become dangerous, and a fearful catastrophe may be the result.

A fact of peculiar pathological significance is, that this disorder is rarely primary in form. There has generally been a previous attack of mania, perhaps of melancholia. The patient has been apparently cured, having become comparatively rational, with the exception perhaps of some one lingering delusion, from which he never recovers. I have had more than eleven years' experience in the treatment of insanity, and have not known a single case of this form of mental disease to be cured. In these cases there has been a cessation of the previous pathological processes, consisting simply in an arrestation of their intensity and perhaps extent. Monomania does not commence as such by any primary pathological change. We know that insanity is always a disease of the brain, but monomania is generally the result of some other form of insanity, in the convalescence from which there has been a clearing up of the morbid condition to a certain point, at which it is arrested, and thus monomania for life remains.

The next form of insanity to which I desire to call your attention is

DEMENTIA.

I have very little to say concerning this affection, having already alluded to it as "the tomb of reason." Some speak of *acute dementia*, as distinguished from *secondary dementia*. By acute dementia is meant that condition which sometimes follows severe fevers, moral shocks, or physical injuries. In these instances the mind, for a time, becomes a perfect blank. I will illustrate this by a case which Professor Dickson, of Philadelphia, was accustomed to relate to his class. A very eminent and erudite divine, of New Jersey, suffered from a severe attack of typhoid fever, from the immediate effects of which he recovered; but his mind was completely wrecked. He had forgotten every-

thing,—could write or speak upon no subject, having ceased to remember not only the elements of arithmetic, but even the alphabet. Being an industrious man, and possessing great tenacity of purpose, he was not overcome by his calamity, but immediately commenced to study energetically, in order to acquire the rudiments of an ordinary education, and thus regain, if possible, all he had lost. One morning, after months had elapsed, the darkness and obscuring clouds with which his mind had been surrounded were suddenly dispersed, the light dawned upon him, and he found himself possessed of all he had lost. It was like the reappearance of the sun after it has been temporarily obscured by a passing cloud.

Asphyxia may also have the same result. I remember being asked once by a lawyer, during a cross-examination, a question very pertinent to the subject we are now studying. A gas company had been sued for damages on account of negligently allowing the escape of gas, which was alleged to have produced acute dementia in an employé, who had been resuscitated from a dangerous state of asphyxia. It was contended by the defense that gas could not have had such an effect, and that the insanity must have pre-existed. The attorney for the company, desirous of showing the similarity of effects between the different forms of asphyxia, many of which he contended were very commonly known and had never been supposed to result in insanity, asked whether I had ever heard of strangulation resulting in mental impairment. I replied in the affirmative. In any variety of strangulation the blood becomes super-carbonized, and necessarily produces deleterious changes in the brain, which may result in some form or other of insanity, perhaps more frequently in acute dementia, damaging the individual very seriously. The lawyer, desiring to propound a perplexing and, as he supposed, absurd question, inquired, "Suppose, doctor, that a man be almost drowned, just saved in time to be revived: do you mean to say that this might result in grave mental impairment?" "Why, of course," I replied; "you have furnished me with an excellent illustration, demonstrating how asphyxia may result in acute dementia, independently of what has caused the asphyxia." This shows the importance of being able to turn their own weapons against those who point them; for many lawyers find no little

satisfaction in making medical men appear ridiculous when called upon the witness-stand.

By chronic or secondary dementia is meant that form which follows the acute forms of insanity, no matter whether mania or melancholia. When you pass through the wards of an insane asylum, you will recognize such patients by their blank countenance. There is an entire absence of intellect, and an incapacity for performing any rational action. You will, perhaps, see them fondly nursing a stick of wood, believing it to be a favorite child, or in the active pursuit of some delusion; in fact, they are reduced to perfect imbecility, and this disease is the gulf into which the various forms of insanity drift. Dementia presents the traces of violent precursory storms, which have stranded the nobler faculties of man, thus animalizing him. Many have not the instincts of beasts, their lives being purely vegetative. Here again you see the necessity of the proper treatment of acute insanity; for what can be more terrible than this condition of abject mental degradation? The neglect and culpability of a physician in not having sent these unfortunate beings, while yet in a curable condition, to an asylum, have to be atoned for by the patients by a life of misery. When they are sent to an asylum they are too frequently hopelessly demented, and the condition to which they are reduced is horrible to witness. They are filthy, often eating their own excrements; and it will excite your sympathy to remember that these people were once rational beings like yourself, and that their fate might have been averted, if only to a degree, by timely assistance. It is an evidence of a flaw somewhere in our social economy.

MORAL INSANITY.

What is moral insanity? Is it an insanity of a man's morals? Is it that condition in which an individual has impairment of mind, destroying his knowledge of right and wrong, or by which he is led to commit nefarious deeds? Is such moral insanity? It is very important to understand this subject, as it is often constituted a plea in legal cases. It is never moral depravity, and moral depravity is not always moral insanity. If you realize this, you have made an important step in advance. In a previous lecture, when I gave Blandford's illustration of an Italian brigand, accustomed to kill and plunder, I showed you distinctly that such

was not moral insanity, but only a blunting of the conscience by the habitual commission of crime. Now, I maintain that moral insanity is but a variety, perhaps in degree a peculiar classification, of the affective type of insanity, of which you have lately heard so much. Affective, impulsive, pathetic, emotional, and moral insanity are virtually one and the same, each and all belonging to the same type. Pritchard, who first described moral insanity, attached great importance to it, especially in a medico-legal point of view. He maintained that in moral insanity there was no evidence, to any degree, of intellectual impairment or implication, and that the judgment, memory, cognition, and perceptive powers were normal, but that there was a perversion of the moral faculties, resulting in a change in the habits, feelings, affections, propensities, and sentiments of an individual, sometimes, though rarely, accompanied by delusions, rendering him insane and irresponsible. He defines it, to use his own words, to be "a morbid perversion of the natural feelings, affections, inclinations, habits, moral dispositions, and natural impulses, without any remarkable disorder or defect of the intellect, or knowing or reasoning faculties, and particularly without any insane illusion or hallucination." I formerly believed this to be true, but am now satisfied that in every case related by Pritchard there was evinced more or less mental weakness or impairment; and while I consider moral insanity to be a perversion of the moral faculties, and am satisfied that every case is accompanied by more or less mental defect, in moral insanity there is more or less (though not apparent) ideational insanity. I have no time to-night to discuss the reasons which have led me to this conclusion.

As for the diagnosis, I must say that I never accept the theory of moral insanity without certain corroborative antecedents of some other form of insanity,—some evidence of the insane temperament, or at least of a strong taint of insanity in the ancestry, whilst I also seek other important links in the history when obtainable. A change in the individual's self is a most important symptomatic manifestation, without which as a basis no case can possibly rest. There must be a change, not to be explained by the ordinary motives of human actions, to constitute moral insanity. Suppose a man is waylaid and killed by some miscreant, the money upon his person stolen, and the thief when prosecuted

should enter a plea of moral insanity to defeat the law: would not the circumstances of the case, and the motive for the act, be sufficient to warrant an expert in insanity in unhesitatingly giving his testimony against such an assumption? But, on the other hand, suppose an upright, honest, moral man, known for a lifetime to be conscientious and above reproach in all his relations,—suppose this man suddenly becomes obscene and lascivious in his conduct, evincing murderous intentions by his acts: if there be superadded an insane ancestry, temperament, or predisposition, we have certainly good evidence of moral insanity. If, however, moral insanity exist, and such a case be investigated, a certain amount of mental weakness will be found, pointing to more or less ideational insanity as a complication of the former. Upon these facts only do I accept moral insanity, but never *per se*, or without some degree of actual mental impairment.

We have now to consider two other forms of insanity:

IDIOCY AND IMBECILITY.

By the former is meant that form of insanity which is the result of an arrestation of development or growth of the brain, either during fetal life or immediately after birth. The latter differs from this only in degree. In it the arrestation of mental development occurs at a period subsequent to birth, often following some pathological process. In point of fact, idiocy is an original defect in the organization of the brain, while in imbecility the defect is only adventitious, and the physical condition of the brain may be perfectly normal at birth, or even at a subsequent period. Still, it is as difficult to draw a line of distinction between idiocy and imbecility as between insanity and reason.

One more form to be considered is

MORAL IMBECILITY.

In mental imbecility the actions express a want of activity of the higher intellect, but in moral imbecility there is an absence of the manifestations of the moral intellect. This is an interesting psychological condition.

Have you never at school seen boys expelled who had no idea of moral obligation, conscience, or the distinctions between *meum* and *tuum*? who were constantly in "hot water," and a source of

anxiety to their parents? In other respects they may have been intelligent scholars, and oftentimes possessed mathematical and mechanical talents, but they were always lying or stealing, and furthermore were given to voluptuous and intemperate tendencies and obscene habits; boys who would forge checks to obtain money; always in trouble while young, and under the eye of the police when grown; although they belonged to good families, and had all the advantages of education and religious training, still they were the black sheep in the fold. You cannot have been close observers if you have not seen such cases, for in every community you may find numerous instances. A boy of this description will be as much trouble to the family physician as to any one else, as the parents will be unable to explain his actions, being averse to thinking him insane, on account of his being so bright in many respects.

Such is moral imbecility. Such people have the misfortune to be born and grow up without the development of those parts of the brain which preside over the elaboration of the moral faculties, and are hence a source of mortification to their relations and a curse to themselves.

Some authors contend that moral imbecility may be congenital, or may be acquired as a result of disease or injuries received during childhood. My friend Dr. William B. Hazard, of this city, has related to me the case of a boy who had always shown excellent moral tendencies until the age of twelve years, when he passed through a severe attack of typhoid fever; after his recovery there was no impairment of the intellectual faculties, but there was such a decided alteration in his morals that his parents were firmly convinced that during his sickness he had become the victim of demoniacal possession.

LECTURE XIV.

INSANITY—*concluded*.

General Paralysis of the Insane.—Three Prominent Characteristics.—Diagnosis.—Illustrations.—Mental Symptoms first developed.—Prognosis.—Pathology.—Treatment.—Diagnosis of Insanity in General.—Acute Meningitis.—Delirium Tremens.—Assumed Mania.—Loss of Memory characteristic of Softening of the Brain.—Prognosis.—Pathology.—Vascular Theory.—Excessive Functional Activity.—Recapitulation.

GENTLEMEN,—In my last lecture I described the principal forms of insanity, sufficiently dilated upon all of them, and gave the symptoms most important to remember. There was one form of insanity, however, which I then had no time to speak of, and which we will now consider. This form is the one generally described in the books as *general paralysis, or paresis of the insane*. The name is really an unfortunate one, as it leads you to believe, as I did before I knew better, that it is a paresis which occurs only in insane persons, or that it is a paralytic condition following insanity, occurring after a certain duration of the latter affection. This is a misapprehension, for many not in asylums are seized with it, and it is an ailment having a tendency to attack persons who are not suffering in any way from insanity. In short, to make it clear, general paralysis of the insane exists in persons attacked *ab initio*, and is not preceded by mania or melancholia. It is a distinct affection, and not amenable to the same laws as mania or melancholia, for one of these diseases may precede or alternate with the other, which is not the case with general paresis, and hence it is recognized by its own characteristic features. The differences are so marked that I do not think it possible to confound them with those of other forms of insanity. Before proceeding further, recollect that it is simply a variety of insanity, may occur in any individual, and may be found in private practice. It therefore behooves you, as physicians, to recognize it, and not to be led astray by its peculiarities. General paralysis of the

insane is very intractable, and its prognosis is gloomy and most unfavorable. Its recognition, therefore, may readily enhance your reputation, while an unfortunate diagnosis would as certainly be a source of mortification. If such a case should happen to come under your observation, you would, of course, extend no hope of recovery. In ninety-nine cases out of a hundred you will find that you have safely ventured upon your prediction, provided your diagnosis be correct. The patient never recovers, and for the simple enunciation of the opinion will you be estimated a very skillful physician. Now, how shall we distinguish this disease?

There are three prominent characteristics enabling you to diagnose it correctly. The clinical features by which it is expressed are so remarkable as frequently to warrant the possibility of an unerring diagnosis. I do not pretend to offer them in the order in which they occur. We have, first, a peculiar paralytic affection of speech, a difficulty of articulation, especially of the consonants. Secondly, there is a peculiar want of muscular co-ordination, which is shown by a remarkably staggering gait, somewhat like that of a man under the influence of liquor. Lastly, there is a strange delusion, consisting of a remarkable exaltation, a curious and overweening self-esteem. A proneness to extraordinary exaggeration and extravagant ideas is rarely absent. Indeed, it seems impossible for the patient to make the least statement without its being greatly exaggerated. These last peculiarities have caused the affection to be termed by the French *manie de grandeur*, or *manie d'exaltation*. Persons afflicted with this disease always entertain a conviction of their own importance and power, and invariably endeavor to impress this upon others with whom they mingle. I will cite you a few instances which happened in my own experience.

I have now under my care a man who labors under the most remarkable self-exaltation. He imagines that he is the wealthiest man in the world, that he actually possesses all the gold in existence. With him money is of no value: he is immensely, incomparably rich, and states that if the waters of the Atlantic were withdrawn and its bed exposed, it could not contain all his gold. He imagines that all the civilized world recognizes in him the greatest living railroad king; and, as he comes from Memphis, he considers that city the great centre of the globe. In its vicinity,

he says, there are immense mountains of iron, which he purchased years ago. This enables him to build railroads in all directions, and he purposes upon them to transport all the larger cities of the United States to Memphis. He has now in course of construction, in Liverpool, immense derricks, by means of which he will raise the entire city of St. Louis into the air and place it upon a line of railroad he is building for the purpose, which will be not merely an air-line road, but three miles, at least, above the surface of the earth. He is quite certain of being able to move our entire metropolis in less than thirty seconds. His resources are inexhaustible. His mountains of silver he claims to have received from God Almighty as an acknowledgment of the fact that he had loaned Him a large sum of money upon some previous occasion. This man is as firmly convinced of the truth of these ludicrous ideas as you, gentlemen, are of your existence. His delusion is the more striking in view of the fact that he is very poor, and not even at liberty, but in an asylum.

An architect under my care some years ago had also a very remarkable idea of his own greatness. He was willing to extend favors to Almighty God, and had in contemplation the construction of a "temple for the world." He had made drawings which were really beautiful and complete in detail. He stated that to this temple all the people of the earth would come to bend the knee. It would be replete with gold, silver, and mosaic; there being, in fact, more of the former than all the nations of the world could supply, he himself having ample means of furnishing inexhaustible quantities. To show you the immense idea of grandeur which had taken possession of this man, he said that on the day of the inauguration of his temple the whole civilized and barbarian world would be present to celebrate the occasion. All of these would occupy the temple, together with all the illustrious dead of past ages, and yet the temple was not to cover more ground than the St. Louis County Court-house! The assembled multitudes would all come to worship the statue of the greatest of living men—himself. His statue was to be of gold, and on an eminence from which it could be seen by all the world. On Christmas-day and the Fourth of July he would mount the eminence in person, so that everybody might adore and venerate him.

Such conceptions are so peculiar and characteristic that a physician of even moderate experience can readily recognize the causative disease. One man will believe that he is the greatest of living generals; another, that he controls the universe, and that upon his care and attention the rotation of the earth upon its axis depends.

It has been a matter of much discussion whether the symptoms of the disease succeed one another in a certain regular order,—whether the paralysis of the organs of speech, or the delusions, precede the difficulty in walking, or the reverse. This question has not been definitively settled, and does not intimately concern you as students. In my own experience, I have generally found the mental symptoms to be first developed, and afterwards the paralytic phenomena, producing the difficulty in articulation and motion. This impediment in speech is more readily observed when the patient is engaged in an animated conversation, and the gait is very similar to that in progressive locomotor ataxia. There seems to be a similar want of muscular co-ordination: the patient does not drag his feet as in hemiplegia, but staggers, and appears to be in danger of falling. An unequal dilatation of the pupils, without being a constant symptom, is rarely absent. Epileptiform and apoplectiform attacks are frequent.

The disease is hopeless, usually terminating in dementia and utter helplessness in two or three years; though some cases are reported of twelve and fifteen years' duration. It is probably a parenchymatous inflammation of the cortical structure of the cerebral hemispheres. The treatment, of course, is negative in its results; and the patient should be confined in an insane asylum.

This completes the description of the principal forms of insanity.

DIAGNOSIS OF INSANITY IN GENERAL.

A few brief remarks in regard to the diagnosis of insanity will now be appropriate. Is there any other affection with which it may be confounded? Is it not possible to make a mistake? I believe that, when fully established, it can usually be recognized by ordinary care and scrutiny. The difficulty is far greater to ascertain its existence when only suspected, especially when called

upon for expert testimony, upon your oath, in a court of law. Some diseases might be confounded with it. Acute meningitis with acute delirium has been mistaken for acute mania; but in the latter there is no elevation of temperature, which is a marked feature of the former; febrile phenomena exist in all inflammatory affections, and are therefore present in meningeal disorders. There is, it is true, an elevation of temperature in one form of insanity, already described, but only in one,—acute delirious mania.

Delirium tremens presents some of the features of insanity, which are manifested when a person has habitually been addicted to alcoholic excesses and prolonged debauches. In ordinary cases of delirium tremens it is hardly possible to make a mistake; because, as you will learn more particularly when we study that disease, there always exists a peculiar loquacity, a remarkable, good-natured delirium, with visual hallucinations, enabling a physician of only slight experience to make the distinction. The tremor also is peculiar, produced, as it is, by the action of the alcohol upon the motor nervous system. But a man may be deeply under alcoholic influence and extremely excited, perhaps with strong homicidal or suicidal tendencies, and the physician may be unable to say whether he is laboring under acute mania or alcoholism. The diagnosis at times may be quite difficult; but we have one fact that may in these cases be of assistance, which is that in ordinary alcoholism the patient is much better after good and refreshing sleep. If, therefore, after having obtained the desired rest, the patient awakes as wild and violent as he was on the day previous, or even more so, the probabilities are that it is a case of acute mania instead of delirium tremens. Besides this, the absence of the ordinary symptoms of alcoholism, namely, the clammy perspiration, the rapid, frequent pulse, and the tremor, readily removes many of the difficulties attending the diagnosis.

To distinguish some particular forms of insanity, and to determine the fact of a man's being sane or insane, are quite different questions, and apt to prove very vexatious. Should you declare a sane person to be a lunatic, and send him to an asylum, thereby unjustly depriving him of his liberty, you may expect that, if he gets out and proves the error of your diagnosis, he will cause you much trouble, especially if you possess any property, which would justify him in instituting civil proceedings

against you. Now, how are you safely to determine these matters, as there is so much difference between the cases we meet? One general rule I can give you in this regard; and that is, always avoid rash and hasty action: never hurry in your decisions, but study the history of the patient, his idiosyncrasies, and in particular any change in character,—an important matter, to which I have alluded again and again throughout my lectures upon insanity. This departure from one's normal self is manifested by certain actions or feelings which never before existed. Be cautious, and do not blindly accept the statements of relatives, as they may be biased in consequence of a desire to remove the person out of their way. You cannot be too prudent; and you should always give the patient the benefit of the doubt. Never blindly assume responsibilities: if the patient be really insane, he will sooner or later show it. If you have carefully followed me in these lectures, you will know that, although a man may suffer from derangement of the affective type with very little evidence of accompanying ideational insanity,—speaking quite coherently and rationally,—he, nevertheless, cannot control his actions; and, as this affective form underlies all others, the actions some time or other are sure to reveal the insanity. If this be the case, the patient must, of course, be isolated by being placed in an asylum, as he is out of harmony with his social sphere, and hence must be sequestered to prevent him from exercising a pernicious influence on the other individuals composing the society of which he formed but a single element. I repeat it, be cautious, be careful, be prudent, before giving an opinion in the affirmative and participating in the subsequent proceedings; for if you do not, you may burden yourself with an immense amount of trouble.

I shall not dwell long upon the differential diagnosis between mania, melancholia, and the other forms of insanity. I only wish once more to impress upon your minds that all insane persons do not necessarily plainly manifest their insanity, and that even experts, after numerous conversations, may be misled, if not upon their guard. If experts have to exercise care, how much more does it behoove the general practitioner to avoid all possible sources of fallacy! If you remember this, you may yet thank me for the stress I lay upon these facts, the knowledge of which

may save many a physician from the annoyance of perplexing dilemmas.

If, as sometimes happens, a delusion exists, and the patient endeavors to deceive you, it may require many visits before you arrive at a knowledge of the true nature of the case, and in matters of this kind you should never allow yourself to be influenced by the desires of friends and thereby be placed in a false position. I have already told you of a case in which an individual so carefully concealed his insanity that, after having him under my care for two years, I felt convinced of his sanity, and consequently wrote to his brother to come and take him home; yet on the very night of the brother's arrival the patient became so violent and aggressive as to necessitate his immediate restraint. Of course, such people are harmless in an asylum; but where they are at large, homicidal tendencies very frequently manifest themselves, as well as other dangerous propensities.

A word concerning assumed mania. You may be called upon to give your opinion in a trial where a man affects insanity in order to evade punishment. You may not have the opportunity of calling in experts, and the criminal will endeavor to deceive you; therefore you must be upon your guard. Recollect that no man can for any great length of time act the part of a maniac. He cannot feign insomnia; for if you watch him or have him watched closely, you will find that in moments of exhaustion he will sleep soundly without the use of an anodyne. Sometimes such people purposely refuse medication. If you have ever seen such cases, you will readily recognize the fact that pretended maniacs invariably overact their parts.

In melancholia, which is a deep derangement of the affective type, we know that, in consequence of a disinclination to take food, the patient will resist its administration. If chronic mania should be simulated, you might be easily misled, and very injuriously, perhaps, to your reputation. But there is one peculiarity in all these simulations of insanity, which the books allude to, that those who attempt the deception always appear to be devoid of memory. They invariably fail to have any recollection or appreciation of time, locality, or condition. These are symptoms more characteristic of softening of the brain than of insanity. I have already told you, in speaking of partial cerebral anæmia,

that there is a loss of memory, which becomes more apparent as the disease progresses. In insanity, on the other hand, there is often the most remarkable remembrance manifested, except in dementia, which, of course, is readily recognized by the expression, or, more correctly, the want of expression, of the face, and by the almost total abolition of the mental faculties, with a previous history of some acute form of insanity. But if mania is feigned these people will often affect to forget that they saw you yesterday, or they do not recollect their own names, or even those of their nearest relatives, etc. We know the absurdity of these assumptions, and the knowledge thereof is of incalculable benefit to the diagnostician. Now, recollect one other important fact in this connection. Insanity in some persons is easily recognized, while in others it is not, nay, is almost impossible of recognition,—even to such an extent that experts will be unsuccessful.

The prognosis can be summed up in a few words. Insanity is curable in direct proportion to the duration of its existence. If acute, it is often amenable to treatment; if chronic, it is not. Therefore, when under three months' duration, it can (in at least ninety per cent. of cases) easily be conquered by therapeutic resources; but when it passes this limit the cure becomes more difficult as the time of duration is prolonged. Again, in patients not over thirty years of age, insanity is quite manageable, but in old people the prognosis is very unfavorable. Where the insane temperament exists, or where insanity is part of the family history, there is less hope of permanent cure. Some of these cases may recover, but a relapse in those who are the victims of an innate vice of constitution or of an instable condition of nervous organization may readily be predicted. Such are always liable to outbursts of insanity, and to have attack after attack; and, if you are acquainted with the histories of cases in asylums, you may easily prognosticate that certain persons will soon return when, being apparently cured, you discharge them. The relapse may be postponed, but you feel assured that, sooner or later, it will occur.

I shall make a few general remarks in regard to the pathology of insanity. All my lectures upon its different forms have been so interspersed with allusions to the pathological origin that there is left but little to add. I have already told you that it is a disease of the brain, and has its seat in definite parts of that

organ,—in the cortical portions of the convolutions of the cerebral hemispheres,—though its initiatory cause may not exist there, but may be remote from it; as in the bladder, uterus, lungs, heart, or other organs. But the proximate seat, the anatomical situation of insanity, is always in the brain. If this be so, do you expect to find after death from insanity manifest changes in the brain? No; for, though it may have been caused by reflex irritation, from the uterus, for instance, which irritation is propagated to the brain, still, anatomical change may not be apparent either to the unassisted eye or to the higher powers of the microscope. But the fact that we cannot ascertain such changes is surely no proof that they do not exist. Just here consider that we are dealing with portions remarkably subtle,—with a labyrinth of remarkable intricacy,—that is, with those parts which lead to the evolution or elaboration of thought. We have not always means sufficiently subtle to detect the molecular changes which may have occurred in this most delicate of all organized structures,—the brain. They may be diseased, and yet the disease may not be apparent to our methods of investigation. Therefore not detecting any change is no proof of its absence. In order that morbid intellectual phenomena may be produced, we must have an alteration in the physiological condition of the cortical cells of the cerebral convolutions, or at least in those that preside over the elaboration of mental phenomena. Dr. Gray, of the Utica Asylum, one of the most gifted and distinguished of American alienists, has greatly contributed to our knowledge of insanity, and has illustrated his contributions to cerebral pathology by means of beautiful micro-photographic illustrations. His conclusions, to quote his own words, are, “Although the cases thus far examined may be regarded insufficient to establish general conclusions, they go to strengthen the conviction sustained by the laws of general pathology, that insanity is a physical disease of the brain, and that the mental phenomena are symptoms; further, that the microscope, with patient and close investigation, will continue to disclose structural changes in the cerebral tissue as marked as those heretofore unsuspected when examinations were limited to the scalpel and naked eye; and in these investigations, when the entire range of the disease, in every stage of its progress, shall have been brought under the microscope, we may

be able to solve the problem of the morbid processes conveniently denominated insanity.

“Another conclusion to which these investigations must naturally lead is, that the variety and changes in the predominant symptoms of insanity may find their explanation not so much in the variety of lesions as in the special parts of the cerebral centres which are morbidly involved in each case; or, to bring the idea within narrower limits, that ideational, emotional, and motor disturbances have their foundation in the extent and degree to which the nerve-elements that minister to the execution of the intellectual and motor acts are involved in the lesion. When the disease reaches its ultimate stage, all distinctions cease, dementia being the same closing stage of every so-called form of insanity.” In the Thirty-Second Annual Report, just published, of the State Lunatic Asylum, Dr. Gray remarks, “Continued experience not only confirms the truth of this position, but also that, in all cases, post-mortem examination will reveal organic lesions, changes in the condition of the vessels or structures of the brain or its membranes.” He discards the term functional disease as a “phantom of the mind,” as it has been designated by Winslow.*

According to the older *humoral* or *vascular* theory, all forms of insanity were considered to be the result of a congestion in some part of the brain. But just as in inflammation there is always a primary tissue-irritation, which is extravascular in the textural components of organic bodies, so in insanity originating from disturbances in the cells themselves, or in the ultimate nervous constituents outside of the vessels, the changes are not primarily due to an increased flow of blood to any parts of the brain. I care not what doctrine of inflammation you have learned, I hold that since the views of Virchow have been promulgated it is an established fact that as in inflammation there is an irritation of the cells composing the tissue outside of the vessels, exactly the same law holds good in insanity, although itself not originally an inflammatory disease, for Dr. Gray has proved that increased connective-tissue proliferation plays an important part as one of its primary factors. Insanity may be superinduced by some adynamic states of the system at times, it is true, through the

* American Journal of Insanity, July, 1875.

influences thereby exercised by means of the blood on the cells and ultimate constituents of the cerebral nervous texture; but the proneness to the disease does not reside in the blood, but in a peculiar instability of the nervous elements themselves or of the cerebral cells, composing as they do the material structure of the temple in which resides the mind. Whenever they experience perturbing influences in their material organization (the bricks and mortar of which the temple is composed), disturbances in the natural evolution of the mental phenomena still residing therein will necessarily occur, the structure being shaken to its foundation, improper ideation will ensue, and insanity will be developed. Therefore the starting-point of all forms of insanity is in the fundamental constituent elements of the little cortical vesicles composing the ganglionic cells of the convolutions of the hemispheres of the brain. Here the disease starts *ab initio*. Hence you will readily see that in insanity it is the constituent elements of these little cells, these minute, almost inconceivable, intangible parts, that are at fault. These may have their own laws of mental elaboration and evolution, of which we know nothing, and which science may, perhaps, never reveal. But how is it with other cells? Do we know the exact working of any of them? Though insanity probably starts in the cerebral cells, it may not be at all improbable that there are consequent though undetectable changes in every other cell in the body, so that an insane person may be insane to the very tips of his fingers. This is surely no more difficult to understand than the fact that the renal or hepatic cells never mistake in the selection of those parts of the blood which are adapted to the manufacture of their proper secretions. Can we explain how aliments are changed into flesh? We know that they are so changed, and accept the undeniable fact of daily transubstantiation during digestion; but can we understand all the particular transformations which occur in these processes of assimilation? Or can we account for cell-impregnation? Why is it that when a woman has children by a second husband they may bear a very remarkable resemblance to the first, though he may have been dead for years? We know that it is so, and might offer an explanation by saying that the cells had been infused by a certain psychical influence which remains for a lifetime; but such an explanation would be at best hypothetical.

But though some of the pathological conditions of insanity are very obscure, there are others that we can more readily conceive. For instance, a man to have a healthy mind must have a healthy body. If his body be pervaded with poisoned blood, whether from syphilis, typhoid fever, variola, etc., it is evident that the functions of the brain will be more or less at fault, because for the production of the healthy evolution of cerebral phenomena we must have healthy blood, which should be normal, both quantitatively and qualitatively. If you ignore this you ignore the fact that a man may be insane with either an anæmic or a hyperæmic condition of the brain. Poisoned blood cannot subserve the proper performance of the actions of the ideational centres. It requires healthy blood to insure the evolution of normal thought, just as in the liver pure blood is requisite for the production of normal bile. You may, perhaps, accuse me of holding materialistic views, because I compare the psychical elimination to that of bile. I must state, however, that the more I study the phenomena of life and the functions of our organism, especially those of the brain, with its wonderful ramifications throughout our system, its ever-varying and almost limitless psychological powers, its delicate mechanism yet mysterious obscurity, the more I am convinced of the illimitable distance betwixt myself, the creature, and my God, the creator. I compare the brain to the æolian harp, whose strings set in vibration by the passing wind give forth euphonious sounds. The mind employs the brain for the production of thought. The mind has no materiality; but if the brain be not in physiological condition its play is in consonant, inharmonious, discordant, and consequently there is insanity. That the brain is only the physical organ of the mind is shown by the fact that the quantity of the phosphates in the urine is in proportion to the amount of mental labor undergone. This is produced by the greater violence of the action of the mind, attended by greater destruction, or retrograde metamorphosis, of brain-tissue.

So much for the manner in which a change in quantity or quality of blood may produce insanity. There are one or more points that still require consideration.

We have seen that insanity has its first origin in the tissues outside of the vessels; but it may be influenced by vascular causes,

and especially by reflex action, which must be relieved in order to effect a cure. I do not think I could give a more beautiful illustration of this fact than one I gave you in a former lecture. It was a case of prolapsus uteri attended by melancholia. This melancholia immediately disappeared upon the reduction of the prolapsed organ; but as soon as the pessary was removed and the uterus once more descended, the insanity reappeared, to vanish again upon a subsequent reduction of the prolapsus. I could not possibly give you a plainer illustration of the effects of reflex action. This is not more difficult to understand than that dizziness and dilatation of the pupil are often symptomatic of the presence of tania, and disappear with the expulsion of the parasite. This proves the necessity of ascertaining the cause of the reflex action, whether it be thoracic, pelvic, or abdominal: direct your remedies to the causative disease, and in relieving this you will often eradicate the insanity.

It is quite a common thing to find females who are insane suffering from metritis, cervicitis, etc.; and the cure of these complications has often restored them to reason. I recollect a case where all previous treatment had failed, in which, after curing an obstinate leucorrhœa, the patient showed the first symptoms of improvement, and finally the insanity disappeared. This shows the connection between cause and effect; and as it is in insanity so it is sometimes in paraplegia caused by reflex irritation from disease of the genito-urinary organs, which is occasionally cured by removing a simple gonorrhœa. If such an affection can have these effects upon the spinal cord, how much more readily will it not act upon the brain, this great centre, this national capital of the body, which is in relation with every tissue, every muscle and nerve and fibre of every part of an organ, and with every cell, part of a cell, or conceivable division of a cell! I do not believe there is a molecule or atom existing in the body which is not more or less under the direct or indirect influence of the brain. Hence a disturbance or perverted influence from any diseased or affected part might produce insanity if kept up for a sufficient length of time.

One more point I shall speak of, and after that I shall dismiss the subject of insanity. Whenever a predisposition to insanity exists, it may be induced by *excessive functional action* of any part,

but especially of the brain itself. This excessive functional activity will produce an irritative exhaustion, and its prolonged influence will result in a disturbance in the part, which being reflected to the brain, insanity follows. Knowing the laws of health, we must try to obviate this, and should never forget that "tired nature's sweet restorer," sleep, which admits of the formation of the recuperative powers and prevents the excessive and incessant wear and tear of the tissues, is the great prophylactic of insanity, and where a prolonged loss of rest has existed, the first thing we must procure is sweet, balmy sleep.

RECAPITULATION.

Insanity, being a disease which has always its seat, though not necessarily its cause, in the brain, is, like other maladies with which the human family is afflicted, divided into the acute and the chronic types. The origin of this sad affliction is not invariably in the brain, as, owing to the close sympathy which exists between this great nervous centre and all the other portions of the body, morbid action starting in any distant part of the economy may, in susceptible individuals, be the exciting cause of a reflex irritation, which deranges the normal action, disturbs the functions, and impairs the delicate mechanism of that wonderful organ whence all the intellectual manifestations are derived.

Too much stress cannot be laid upon the perfect realization of the fact that insanity is curable in direct ratio with its duration. Although, exceptionally, cases of many years' continuance are sometimes restored, and notwithstanding the well-admitted fact that no case is necessarily incurable, yet for all practical purposes it is in the field of acute cases that the physician expects to reap the most abundant and successful harvest. Many authors place the limit of the acute stage at three months from the inception of the derangement. The farther we pass the third month, the more difficult of accomplishment will be the cure. You are all doubtless aware that prognostications of ordinary pulmonary affections are favorable just in proportion to their recent or remote origin. Pneumonia, or acute inflammation of the lungs, with modern therapeutic resources, is not often a fatal disease. Phthisis pulmonalis, or consumption, still but too often baffles the best-directed efforts to master its terrible ravages. On the

other hand, the management of cases of acute mania or melancholia frequently attests the brilliant results accomplished by psychological physicians, whilst monomania, chronic mania, and dementia are still the opprobrium of our art. Dementia is comparable to a battle-field, where once all was storm, fury, overwhelming and irresistible violence, but now all is wreck, ruin, and desolation. The heyday for the multitudes of unfortunate victims has passed forever, and the salient fact which fills us with regret is that this unfortunate result remains, because they did not reach the haven of the asylum in time to prevent the disaster from which they will never recuperate. The irreparable mischief has been already achieved through procrastination of friends and faulty and unphilosophical legislation. The conclusion to be drawn is manifest,—send all patients to the asylum upon the very first development of mental aberration; and the successful treatment which will follow such a procedure will soon convince you of the wisdom of this course. The morbid processes, if you lose valuable time, make sure and rapid strides, and every day that you allow to pass without medical treatment will but aid in the confirmation of the terrible doom which awaits those whose misfortune has brought upon them the curse of chronic insanity.

LECTURE XV.

APOPLEXY (CEREBRAL HEMORRHAGE).

Cerebral Hemorrhage: Causation.—White Apoplexy: Diagnosis.—Differential Diagnosis: Coma, Syncope, Asphyxia, Epilepsy, Alcoholic Narcotism, Opium Narcotism, Meningitis, Arterial Obstruction, Sunstroke, Uræmic Coma.—Precursory Signs of Apoplexy.—Modes of Seizure.—Encephalitis.—Prognosis.—Post-mortem Appearances.—Hemorrhage of the Cerebral Membranes.—Hæmatoma of the Dura Mater.

GENTLEMEN,—To-night I purpose considering an affection generally known as apoplexy. The word is derived from the Greek *απο*, from, and *πλησσω*, I strike. It means an instantaneous seizure or fit of unconsciousness, or sudden attack of coma. Those who were present when I lectured upon hyperæmia of the brain will probably recollect that I stated the word apoplexy to be really an unfortunate name or definition, usually applied to, and used synonymously with, cerebral hemorrhage. Apoplexy literally means certain phenomena characterizing conditions common to many different pathological states. The term cerebral hemorrhage is undoubtedly preferable to designate the particular malady which is the subject of to-night's lecture. The former, however, is the term still in general use, having been adopted almost by common consent, though, as I have already stated, simply expressing a certain comatose state incident to many diseases, as we shall soon learn. Then, as Trousseau remarks, such a sudden and unexpected seizure is not characteristic of cerebral hemorrhage, which supervenes more gradually than is generally supposed; a fact upon which medical writers do not sufficiently dwell.

There are always well-defined premonitory symptoms, which, to the physician, point out the coming storm, and precede the unconsciousness, the loss of voluntary motion and of sensation, which constitutes the true apoplectic state. The name, therefore, is evidently a misnomer, and although in a measure compelled to adhere

to it on account of its adoption by most authors, I shall nevertheless use the term cerebral hemorrhage as often as possible.

In order to be well understood, and at the risk of becoming tiresome, I desire once more to state that the word apoplexy refers in reality to a condition common to various pathological crises,—apoplectic symptoms occurring in hyperæmia as well as in anæmia of the brain, also in epilepsy, alcoholism, cerebral hemorrhage, and uræmia. Coma is synonymous with the term apoplectic phenomena.

As regards *cerebral hemorrhage*, have the popular beliefs any foundation? Are persons of a full habit, with short necks and protuberant bellies, more liable to attacks than others? Upon this subject authors do not agree. But let us inquire into the different modes of *causation* of cerebral hemorrhage, and ascertain the ordinary factors in its production. It is a well-known fact that many of the active causes of hyperæmia of the brain may result in this malady. An increased action of the left ventricle of the heart, a diseased condition of the arterial blood-vessels, produced by *endo-arteritis deformans* (upon which I have dwelt in former lectures), followed by atheromatous changes, rendering the arteries more friable and brittle and liable to rupture through excitement or powerful emotions, may lead to cerebral hemorrhage. This morbid state of the arteries, as I have previously told you, generally occurs in persons who have passed the meridian of life. Children, however, have perished from cerebral hemorrhage; though such an occurrence is rare.

Disease of the kidneys may result in cerebral hemorrhage, as follows. The kidneys are the principal means by which certain excrementitious substances are eliminated from the blood; and should their action be defective, these deleterious matters, instead of being removed, accumulate, poisoning the blood; and all the tissues of the body thereby nourished will become more or less contaminated. The nervous tissues are the first to feel the noxious influence and rebel against it, being more peculiarly susceptible. The *materies morbi* of gout, rheumatism, and some other blood-poisons will lead to pathological changes in certain organs, and also sometimes induce renal disease, necessarily resulting in irritation of these nervous tissues, and as a consequence a certain change in their histological constitution. No longer replenished by

healthy blood, a morbid condition ensues, and the coats of the cerebral arteries—more perhaps than of the other arteries—become affected with a fatty degeneration; and in the event of any subsequent affection of the brain these arteries are more liable to rupture, and all this train of morbid phenomena is produced in consequence of the primary pathological condition of those distant organs, the kidneys.

You can easily perceive, therefore, the manner in which a disease of a remote part may produce cerebral hemorrhage: it may be the heart, or there may be a primary change in the blood-vessels themselves, independent of any influence of the factors above mentioned. We can readily understand under such circumstances that physical causes, whether exciting or depressing, might occasion a rupture of the vessels and produce cerebral hemorrhage. The disease of the vessels is the predisposing, and the physical or moral influences are the exciting, cause.

White or serous apoplexy is an effusion due to a collateral œdema, the result of a hyperæmia, or of an anæmia with collateral hyperæmia and subsequent œdema; it is generally produced in the subarachnoidean spaces, or in the meshes of the pia mater, or in the ventricles by a transudation of the serum of the blood. Bear in mind that we have apoplectic phenomena sometimes without extravasation of blood, the result of the ordinary condition described as *ictus sanguinis*, or blood-stroke.

I have now given you the principal facts concerning the pathology of the disease. It would be idle for me to describe certain cases and expect you to tell by certain symptoms whether or not we have to deal with cerebral hemorrhage. My reason for expatiating upon this subject at length is owing to the misappellation “apoplexy,” which is so liable to lead to confusion, relating as it does to the apoplectic phenomena occurring in numerous varieties of pathological conditions having the most diverse origin. I shall take up many of them seriatim and show you how easily they may be mistaken one for the other.

It is evident that, as the apoplectic phenomena are common to all forms of coma, the physician cannot possibly determine the nature of the disease simply by these manifestations. But he must do so by the history of the case, and by certain other considerations, hereafter to be entertained and explained. The question is

one of the utmost importance, and should be decided with great circumspection.

It has been my custom heretofore to give two lectures upon *coma*, but, not having sufficient time this year, I must necessarily condense a great deal. I shall, however, embrace in this *differential diagnosis* all the principal diseases in which apoplectic symptoms exist, and which might be mistaken for cerebral hemorrhage. Let me first give you a definition of apoplectic phenomena. By these I mean a sudden cessation of consciousness, of sensation, and of voluntary motion. If you have ever seen any one faint, you must have been struck with the similarity of the symptoms to those of apoplexy. The patient falls, becomes pale, is more or less unconscious, and nauseated, with an apparent abolition of voluntary motion, etc. Hence you might mistake a simple *syncope* for a cerebral hemorrhage, which would be a very serious blunder, especially should you have recourse to active treatment, as is recommended by the older writers in the latter disease. Such disastrous errors can be avoided by taking into consideration the extreme pallor of the face in syncope, and the facility with which a return to consciousness and voluntary motion is brought about by the administration of stimulants and lowering the head.

The following illustration will make it apparent that I have not given an exaggerated opinion of the danger of making mistakes in these cases. Dr. Stokes, of Dublin, relates that once he had a patient subject to habitual attacks of syncope, owing to some aortic valvular lesion. The doctor generally arrested these attacks by the timely administration of an ammoniacal stimulant; or the patient, feeling the approach of the attack, would place himself on his hands and knees, and the tendency to syncope would quickly pass off. But once it happened that the doctor was absent from the city when his patient was seized with his usual attack, and another physician who was called to the sufferer declared the attack to be one of apoplexy, treated him actively, and nearly sent him to an untimely grave. An important symptom in these cases always is the condition of the pulse. In cerebral hemorrhage this is usually full, slow, and labored, while in syncope it is feeble and frequent.

The next condition is *asphyria*, which should you ever have witnessed you will clearly remember, the semi-apoplectic symp-

toms of which are due to the deficiency of oxygen and the carbonization of the blood, in consequence of which the functions of the brain are impeded. How are we to detect this? First, by the history, which is of the utmost importance in all cases, whether the disease be of the heart, lungs, or any other part. In asphyxia the history will generally be evident, and prevent a mistake. The peculiar lividity of the mucous membranes, and the cyanosed hue of the skin, are significant symptoms.

The next form is epilepsy, to which disease I have already adverted on several occasions. We will suppose a patient to fall into an epileptic attack in this amphitheatre. We would first have convulsive phenomena, but these would soon disappear, and would be succeeded by profound sleep,—a comatose state,—lasting perhaps half an hour or more. But suppose the entire faculty of this city to be summoned just at this comatose stage of the case, could they, if not acquainted with its history, the origin or mode of invasion of the attack, rapidly make a correct diagnosis?—finding only one set of symptoms present, namely, an absence of consciousness, stertorous breathing, and abolition of sensation and voluntary motion? No, not by any means; an opinion under such circumstances would be very hazardous. Upon inquiry, however, should they learn that the patient had at times been subject to epileptic seizures, they would, of course, assume the case to be probably one of epilepsy, and look for an early return of consciousness. You once more see the importance of obtaining a history of the case, since it is absolutely impossible to jump at a diagnosis without some data to start from. And let me caution you always to be extremely prudent in giving a definite opinion in an emergency of this description. Suppose a patient to be in the comatose condition just described. Dr. A. comes in, examines him, and very gravely tells the relatives that it is apoplexy, and the patient will in all probability die, or that he will be paralytic for several months,—perhaps the remainder of his life. Dr. C. then arrives, inquires into the previous history of the patient, etc., ascertains that it must be a case of epilepsy, and reports that the patient will soon recover, with no paralysis, or with a paralytic condition which will pass off in a few days. In point of fact, it happens that in about half an hour the patient gets up, walks about apparently well, and the doctor of grave prognosis is in an ugly predicament:

having left no loop-hole through which to escape, he is compelled to regret the consequences of a hasty opinion. These are not cases for specialists, occurring as they frequently do in private practice. It is not an uncommon thing, under such circumstances, to be called out at night to find everybody alarmed, friends not knowing what is the matter, doctors present from all quarters, and more looked for,—all anxiety and excitement. Let me warn you once more, if you do not know with certainty what is ailing your patient, whilst not manifesting your ignorance, do not injudiciously commit yourselves.

What other conditions are there presenting apoplectic phenomena? *Narcotism* is one to be mentioned, which occurs in different forms. You have, perhaps, seen persons in a profound stupor from beastly intoxication. This is *alcoholic narcotism*. A true apoplectic state exists,—the breathing is stertorous, the patient is unable to move, and absolutely unconscious. Wherever he may fall there he will remain, regardless of the locality or of the efforts of those around him. He cannot be aroused: his stupor is deep, and his state one of utter helplessness. It is not always easy to differentiate between this condition and cerebral hemorrhage, and misapprehensions but too often occur in this respect. It is related by good authorities—Tanner and Watson, for example—that the police of London frequently arrest and take to the station-house persons supposed to be drunk, but who in fact are suffering from apoplexy. Suppose your father or brother the victim of such seeming stupidity on the part of the policeman, whose action had perhaps killed your relative. You would, of course, be very indignant; but when you reflect that physicians themselves make identical mistakes, you will not so hastily blame these officers of the law. Should you be in doubt in diagnosing such cases, you must patiently await developments. I admire the tact of the physician who knows how to keep his own counsel under similar circumstances, instead of compromising his reputation. The best diagnosticians in the world are liable to error.

The apoplectic condition caused by drunkenness may generally be recognized by the presence of a strong alcoholic breath. A positive odor of whisky, brandy, or gin will make you cognizant of the true state of affairs. The best remedy then is ammonia; but

beware of, and prepare the bystanders for, the sudden and wonderful results from its administration. The rapid restoration to consciousness (or activity, rather) seems almost incredible: a violent delirium makes it advisable for a short time to keep at a respectful distance, to avoid a kick or a blow. Why this state should be developed I do not know; neither can I positively explain the mode in which ammonia disperses the coma; though it may perhaps be that it forms with the hydro-carbon of the alcohol a carbonate of ammonium, which, in turn, is eliminated by the lungs or other emunctories. I can only tell you positively, in regard to its action, that it generally rapidly sobers profoundly intoxicated persons.

But in narcotism due to *opium*, *belladonna*, or any other of the narcotic poisons, where the same stertorous breathing and unconsciousness are found, how shall we determine the cause of the resulting phenomena? It is often extremely difficult; though in narcotism from vegetable poisons there is usually a marked dilatation or contraction of the pupils. Such cases are of frequent occurrence. I was once called, with other physicians, to examine an old gentleman who was in an apoplectic condition, and, as we had no medical history, we could not determine positively whether there was a clot in his brain, or laudanum in his stomach, since a half-empty bottle of the drug named stood on the mantel-piece. The patient died without a determination on our part of the cause which produced the apoplectic condition. This instance will show you how perplexing it often is to make a diagnosis in cases where coma is the only symptom,—a state common to so many very different diseases. If, in the above case, one of the physicians had concluded upon the existence of a clot in the brain, due to cerebral hemorrhage, and another had used flagellation or applied electricity, causing the patient to open his eyes, get up, and walk about, the former would have been placed in a most mortifying position.

We have not yet considered all the forms of disease in which coma may be present. *Meningitis* may terminate in coma; and how are we then to differentiate? Fortunately, we have some indication here at least that will assist us, unless we be rash and careless. Meningitis, you know, is an inflammatory affection, accompanied always by fever, headache, and vomiting. But, you

may ask, can we always discriminate between meningitis and cerebral hemorrhage? In ninety-nine cases out of one hundred we can; though not in exceptional cases, as, for example, in *méningite foudroyante*, the siderant form of cerebro-spinal meningitis. I have told you that in these cases a patient may die in five hours; that you may find him comatose at your first visit and dead at the second. A mistake here is possible; yet the known presence of the epidemic influence of the disease should make you avoid a faulty diagnosis.

The next form of apoplectic phenomena which might lead you into error is *arterial obstruction*. In my lectures on anæmia of the brain, I told you that a clot plugging the Sylvian or middle cerebral artery would cause the manifestation of apoplectic phenomena,—loss of consciousness, of voluntary motion, etc. You may ask, Is this cerebral hemorrhage, or is it embolism? You may perhaps determine by the precursory signs of embolism. I have told you that in the diagnosis of all cerebral troubles you should never fail to explore the chest, and a cardiac disease revealed by murmurs, with a history of rheumatism, or a destructive disease of the lungs, will point to embolism and anæmia of the brain, and not to a clot or cerebral hemorrhage. For the full particulars of this diagnosis I refer you to the description I gave you of *anæmia and hyperæmia of the brain*.

The next form I shall speak of is *sunstroke*, with which affection you may meet every summer. The causation of the apoplectic phenomena of sunstroke is generally to be ascertained by the history of the case and the existence of extreme heat, and by the occurrence of other cases of this kind. As I have told you, the apoplectic pulse is full, slow, and labored; whilst that of sunstroke is generally feeble and frequent. In sunstroke there is also greater difficulty of deglutition than in cerebral hemorrhage; and the pallor, the respiration (less labored than in cerebral hemorrhage), and especially the pulse, are the most trustworthy symptoms. I neglected to tell you that in narcotism, whether due to alcohol or to opium, a person may sometimes be temporarily restored to consciousness by violent shaking or by flagellation; though, when this is discontinued, he will almost immediately relapse into coma, which is not the case in cerebral hemorrhage.

The last form for consideration is *uræmic coma*, the apo-

plectic phenomena of which disease are brain-symptoms, complicating some variety of the kidney-affections known by the collective name of Bright's disease; and in the affection characterized by the small, contracted kidney, we often find the uræmic symptoms produced. The blood is poisoned, the nutrition of the brain perverted, and coma supervenes. But here we invariably have a primary history of toxæmia. We find, first, a diminution of the urine; then a convulsive seizure, like an epileptic attack, preceded by vomiting. This alone ought to distinguish it from cerebral hemorrhage. After the convulsions coma sets in, becoming deeper and deeper, and the patient dies, unless the repeated efforts to restore the flow of urine prove successful. Should you be in doubt as to the nature of the case, there is nothing simpler than the introduction of the catheter into the bladder; and should only a few drops of urine be drawn, you can test this for albumen, the presence of which will oftentimes be sufficient evidence of disease of the kidneys, with a resulting uræmic coma.

I shall now proceed to enumerate some of the different *signs* preceding attacks of cerebral hemorrhage. There are usually premonitions, such as headache, aphasia, difficult deglutition, scintillations before the eyes, dizziness, with motor and sensory disturbances. Such symptoms may exist in hyperæmia, or may be precursory signs of cerebral hemorrhage. Persons are never stricken suddenly, but there will have been some characteristic warning, such as a peculiar numbness of the limbs, half a day or more preceding the attack; and this numbness is generally on that side of the body which is about to be paralyzed. Following this is the seizure,—the coma,—and the person is felled like the ox by the axe of the butcher, after which occur the phenomena common to all the varieties of diseases we have considered to-night,—epilepsy, uræmic coma, narcotism, etc. The phenomena presenting themselves during the coma are profound sleep, slow and laborious breathing; the velum palati and the buccinator muscles are paralyzed, and the cheeks audibly flap like sails in the wind,—this last feature, however, being more common in the coma of cerebral hemorrhage than in the others. The pupils are dilated; the patient cannot be aroused; the pulse is slow and labored, beating about forty or fifty to the minute. This picture

is surely sufficiently graphic to insure the recognition of the affection. Finally, the patient, if beyond the reach of medical skill, dies,—or, if not, is restored to consciousness with more or less paralysis, and with a prospect for a second, or even a third, attack. He may remain hemiplegic during life, as is generally the case; though it sometimes happens that the paralysis partially disappears. It is permanent and extensive in proportion to the size of the clot and the parts of the brain which are involved.

There remains one more fact to which I wish to call your attention: namely, that sometimes, three or four days after an apoplectic attack, the patient becomes feverish, with a rapid pulse, burning skin, flushed face, and injected conjunctiva. These are symptoms of irritation, and are indicative of an inflammation of the brain-substance surrounding the clot,—a reactionary period, in which we find a partial *cerebritis* or *encephalitis*, which is generally limited or circumscribed.

The *prognosis* of cerebral hemorrhage is not very encouraging. Some get well, but many die. It is a very fatal disease, and if the patient survives the first attack he will be liable to others.

The *post-mortem appearances* are extravasation of blood and a clot in the brain (this, however, is not the case in white apoplexy). The clot is usually found in the neighborhood of the thalamus opticus or the corpus striatum, or in the substance of the cerebral hemisphere, the crura cerebri, the pons varolii, or the medulla oblongata. These situations are given in the order of frequency of the probable location of the clot. Where the clot is in the region of the pons varolii, or the medulla oblongata, or the corpora quadrigemina, the trouble is vastly more serious than where the medullary portion is affected. It is upon the particular locality of the brain which is affected, and the amount of tissue which is ploughed up and disintegrated, that the gravity of the symptoms depends. Should the parts involved be those which preside over the innervation of important organs, as those of circulation or of respiration, the consequences will necessarily be fatal. The clot may either be re-absorbed, or may become encapsulated by a delicate membrane. Finally, it shrinks, and in proportion to this shrinking is the subsequent damage smaller and recovery easier.

HEMORRHAGE OF THE CEREBRAL MEMBRANES.

Extravasation of blood in the subarachnoid spaces is frequently of traumatic origin. Oftentimes the resulting symptoms do not manifest themselves for some hours, due to the gradual escape of blood from some ruptured artery. This is very significant in a diagnostic point of view, and you will frequently meet with cases of severe head-injury where but few indications of trouble occur for twenty-four hours, when the development of cephalalgia and gradually increasing coma, accompanied or unaccompanied by epileptiform convulsions and paralysis, will unerringly point to the nature of the mischief which has been accomplished. This form of hemorrhage may also be due to injury of the head during birth and in new-born children, to the rupturing of diseased vessels, bursting of aneurismal dilatations, or in consequence of the giving way of one of the sinuses of the dura mater.

The extravasation sometimes occurs between the dura mater and the arachnoid, and in some instances it may extend to the ventricles. The hemorrhage is necessarily diffuse, and the resulting symptoms are not regional. All authors agree in the statement that the apoplectic phenomena are extremely profound, and that this form is quite fatal in its results. When patients die suddenly, without premonition, we have what has been termed "*apoplexie foudroyante*." Hemiplegia is usually absent, as the hemorrhage is not limited or restricted in character, and when paralysis occurs we would necessarily expect it to be general. In all diseases affecting the convexity of the cerebral convolutions, epileptiform convulsions are common. These latter, co-existing with profound coma and the absence of hemiplegia, constitute the diagnostic points of meningeal hemorrhage.

HÆMATOMA OF THE DURA MATER.

"Sanguineous flattened masses, composed of fine layers of fibrin, spread to a greater or less extent over the dura mater, accompanied by small extravasations, which are converted into pigment. By repetition of the process numerous layers come to be deposited one upon the other. Numerous and large blood-vessels form in these layers, and from these vessels renewals of the hemorrhage

occur. The disease is chronic, and terminates, after continued cephalic suffering, generally suddenly, with symptoms of apoplexy."—Aitken.

Niemeyer holds that the disease is more common in old people, and particularly so among drunkards and the insane. Its origin is sometimes traced to "injuries of the brow." "In the latter case, years, it is said, may intervene between the injury and the first symptoms of hæmatoma." Its location is nearly always in the neighborhood of the sagittal suture, in the form of an "oval flat sac." Niemeyer, quoting Griesinger, states the following facts connected with the consideration of the diagnosis: "If the circumscribed headaches, gradually increasing to great severity, in the vicinity of the vertex and of the forehead, be the first, and, for a long time, the only, trouble of which the patient complains; and if, between the appearance of these pains and that of other severe brain-symptoms, there be an interval not so short as in acute diseases of the brain and its membranes, but shorter than in most chronic diseases of these parts, particularly in the different cerebral tumors, the first suspicion falls on inflammation of the meninges, particularly of the dura mater, since inflammation of the other membranes has so great a tendency to spread that it is accompanied by diffuse, not by circumscribed, headache." The term *pachy-meningitis interna* has been applied to this disease, in contradistinction to *pachy-meningitis externa*, which we described in a former lecture.

The hæmatoma is usually from four to five inches long by two and a half broad and half an inch thick. The tumor is generally unilateral, but sometimes is bilateral. It is a disease which occurs after the individual has passed the meridian of life. Finally, symptoms of depression follow the interval usual after the initiatory symptoms of irritation have lasted for some time, in which latter stage we find violent and localized cephalalgia as an almost pathognomonic manifestation. The psychical functions are depressed, the memory and general intellect impaired. This is undoubtedly the result of the pressure upon the convolutions of the brain attended by consecutive anæmia or softening, occasioned by the hæmatoma. Somnolence, with a gradually increasing tendency to coma, becomes more and more developed. Occasional attacks of transitory unconsciousness are produced towards the termina-

tion of the affection, with the development of a partial hemiplegia.

Griesinger attaches importance to almost constant contraction of the pupil, which he regards as a symptom "of irritation of the surface." Aitken considers the possible antecedent existence of syphilis a factor not to be ignored in cases of hæmatoma of the dura mater.

LECTURE XVI.

THE DIAGNOSIS AND TREATMENT OF CEREBRAL AND MENINGEAL HEMORRHAGE.

GENTLEMEN,—We have seen during the previous lecture that the term apoplexy is oftentimes so unfortunately associated with the term cerebral hemorrhage as to produce inextricable confusion, by confounding symptoms resulting from several very distinct pathological conditions having but one and the same causation. It will therefore be of importance to recapitulate the various affections in which *apoplectic symptoms* are of the most frequent occurrence,—leading, perhaps, to errors in the diagnosis,—before we attempt to discuss the treatment of cerebral hemorrhage.

We now approach the differential diagnosis of affections in which *true* apoplectic symptoms are found accompanied with *paralysis*, and with which we must familiarize ourselves in order to diagnosticate correctly by exclusion.

It is of such common occurrence for medical men to confound these cases with those of cerebral hemorrhage, because of the presence of apoplectic symptoms, that we cannot too carefully appreciate the various sources of error which may induce us to treat distinct pathological manifestations, presenting precisely analogous symptoms, by similar methods of treatment, antagonistic to the welfare of the patient.

To illustrate this, I may mention that many forms of hyperæmia and anæmia of the brain produce the same symptoms; and every tyro in medicine is well aware of the great importance attached to the necessary distinctions in the treatment of two such opposite pathological conditions. It is only by basing our diagnosis on the etiology, the sequence of the symptoms, the history of the case, and the age of the patient, that we can arrive at definite and reliable conclusions.

The two principal affections presenting apoplectic symptoms

accompanied with paralysis to be guarded against in the diagnosis of cerebral hemorrhage are, first, acute or red softening; secondly, atrophic or white softening (brain-necrosis of Niemeyer), especially that produced by obstruction of the cerebral arteries with emboli, which, by the sudden anæmia of the brain it occasions, produces the symptoms under consideration. In the diagnosis of acute or red softening, we must recollect that the latter is the result of a previous encephalitis.

The differential symptoms from those of cerebral hemorrhage may in some cases be impossible to determine; in many cases, however, the gradual manifestation of symptoms of psychical depression and slight motor and sensory impairments, in connection with the etiology of the case, will determine the diagnosis. Durand-Fardel mentions as "a significant sign of acute softening, an increased secretion from the mouth and eye."

As regards the second point,—the distinction of the apoplectic symptoms caused by obstruction of an important cerebral artery, whereby a large territory of nervous substance is suddenly deprived of its nutritive supply,—a careful auscultation of the heart and lungs will be the keystone on which our diagnosis can rest with great security. If, during the course of an endocarditis, or in the case of a patient suffering with valvular disease or an extensive pulmonary destructive affection, such an accident occurs, our attention will be immediately directed to the probability of an embolus, occasioning obstruction in an important cerebral artery. Hence it behooves prudent men to explore the thoracic cavity in all cases of so-called apoplexy with the same diligence that they would give to the examination of the urine in chronic brain-diseases; knowing how, link by link in the pathological order of sequences, a diseased condition of the nervous centres may have originated from an impoverished and poisoned nutrition, the result of a want of proper elimination of excrementitious substances retained in the blood through the faulty and inadequate performance of the renal secretion, occasioned by serious degeneration of the kidneys.

We are now ready for the following law, almost invariable in its application to the differential diagnosis of atrophic softening and cerebral hemorrhage, first enunciated by Récamier, and quoted by Trousseau: "That whenever hemiplegia, complete and absolute,

occurs *suddenly*, without loss of consciousness, softening of the brain may be diagnosed. Whenever, on the contrary, the complete loss of motor power is attended by loss of consciousness, whenever especially the individual has become *suddenly* comatose, hemorrhage may be diagnosed, and hemorrhage to a considerable amount.

"But when the intellect is affected to some extent, but not entirely; when there is obtuseness, but not complete loss of sensibility, whilst there is absolute loss of motor power, we must always, according to Récamier, diagnose hemorrhage, in connection with softening, or what has been termed capillary hemorrhage."

In softening, the sudden occurrence of paralysis is due to an abrupt giving way of the continuity of the brain-fibres, producing an interruption to the conveyance of the mandates of the will from the centre of volition along the motor tract.

It may here be important to allude to the fact that in cerebral hemorrhage, far from a sudden and unexpected development of apoplectic symptoms, such as is generally supposed to occur, there are almost invariably certain prodromata plainly indicative of the approaching storm. Trousseau, with his vast experience, attests, "For more than fifteen years my attention has been directed to this point in the history of cerebral hemorrhage, and I never had the chance, *never once*, of seeing a patient struck down *suddenly* by apoplexy, in the classical and etymological sense of the word. I have indeed seen a great number of individuals suffering from cerebral hemorrhage, in the most profound apoplectic stupor, but in every case, *without exception*, when the attack had occurred in the presence of witnesses, it had come on gradually, and had in general been slight at the onset, coma supervening ten minutes, half an hour, an hour, or several hours afterwards; but in no single instance, I repeat, have I seen a man with cerebral hemorrhage struck down as with a blow, and dropping instantly in a state of unconsciousness." Hence the *gradual* occurrence of cerebral hemorrhage and its appropriate prodromata are elements not to be forgotten in the diagnosis, constituting, as they do, characteristic features of the affection. This brings us to the discussion of the treatment,—a matter of the most serious import to the practitioner.

After a careful but concise review of the teachings of the prin-

cipal modern schools of medicine on the subject, we shall try and cull from them all that constitutes the most reasonable and judicious measures, avoiding such as will not bear the severe test of reflection and common sense. As exponents of the different schools, we have selected Todd, J. Hughes Bennett, J. Russell Reynolds, and Tanner, of the British, Flint of the American, Trousseau of the French, and Niemeyer of the German. If any partiality has been evinced in this selection, we must plead as our apology the high authority in which they are held in their respective countries, the encomiums they have received from the profession at large, and the clearness, decision, and plausibility of their opinions.

Bennett regards the measures to be followed during the attack as "a subject of anxious consideration." He says,—

"The best rule, therefore, I can give you, is to judge of all the circumstances of the case. Whenever the individual is of a vigorous frame of body, if the face be flushed, the attack recent, and the pulse strong and full, a moderate bleeding may be beneficial. The extent must be influenced by its effect upon the heart's action; for, as we have seen, the object of the measure is not to draw blood from the brain, which is impossible, but to diminish the pressure on that organ by lessening the force with which the heart propels blood through the carotid and vertebral arteries. On the other hand, if the individual be of spare habit, the face pale, the pulse weak and irregular, and the usual symptoms of shock be present, wine, brandy, stimulants generally, and restoratives, are demanded. But it most frequently happens that when you are called in, neither one nor the other indication presents itself. It will be most prudent, under such circumstances, simply to apply cold to the head, administer an active purgative, and, above all, enjoin quietude. At the same time, the patient should be placed in the horizontal posture, with the head slightly elevated, whilst the cravat, stays, and all impediments to the respiratory and circulating functions should be removed."

This judicious advice is a gentle admonition to those who consider the lancet the *sine qua non* of proper treatment, reminding them that active measures may in many cases be more powerful for evil than for good, and that a more enlightened pathology narrows the limit of the usefulness of venesection. Tanner

premises his remarks upon the treatment of the fit by the following statement: "*Formerly the treatment of every case of apoplexy was commenced by bleeding, and statistics prove that the more freely the blood was taken away, the greater was the mortality.*" In his comments upon this assertion, he adds that such results might be expected, inasmuch as the patient is seen only when the damage is done, and bleeding will not remove a clot. He alludes to the difficulty of determining in such instances the previous condition of the arteries, the prodromata of the fit, and the state of the brain itself. In case of disease of the arteries, or of previous renal or cardiac disease, he asks whether venesection would not be followed by unfortunate consequences. He recalls the fact that the reparative processes could not be aided by such a mode of treatment. He then meets the argument that depletion will prevent further extravasation by the statement that, on the contrary, it promotes it, "*partly by diminishing the power of coagulation, and partly by inducing greater thinness of the blood.*" He quotes Mr. Copeman in support of this same view. The basis of his treatment consists in obviating the tendency to death, and only practicing a small blood-letting in cases where the tendency is to death by coma, the pulse full, hard, or thrilling, the vessels of the head congested, the face flushed and torpid. In the article on apoplexy in J. Russell Reynolds's "System of Medicine," we read as follows: "The recovery of a patient depends, doubtless, altogether on the quantity and seat of the sanguineous effusion, *and not on our treatment.*" "If the ventricle be opened, if a large clot be in the pons, the patient will die; but, as we cannot be absolutely sure that the patient has got a clot in his brain at all, we must do all we can for coma which may be due (besides drunkenness, poison, and injury) to uræmia. There is, unfortunately, little to be done. The chief thing is to keep the patient quiet." Speaking of the supposed advantages of bleeding in certain cases in which it might be cautiously performed, he adds, "It may be that bleeding from the arm will serve the patient, yet I confess to having no great hope of its being of value to relieve the embarrassment of the venous circulation of the patient whilst the cause of the embarrassment still keeps its place inside the head. Indeed, my faith is so little that I have never adopted the practice. But we know that a few patients come out of deep coma with embar-

raised respiration, and it is possible that bleeding might help more out of it." A little afterwards, he adds, "So far as I have observed, however, the practice in this country must be exceedingly rare, as I have in the whole course of my life seen but one person bled for cerebral hemorrhage." "The more vividly we realize the fact that a mass of blood is lying abroad in softened and true nervous tissue, the less confident do we feel in our personal power to interfere. *There is, to speak shortly, to my knowledge, no treatment for effusion of blood in nervous tissue.*"

Flint expresses his views of venesection in apoplexy as follows:

"In the treatment of cases of apoplexy, an important question is, whether blood-letting shall be employed. If the apoplexy be dependent upon active congestion, and the heart acts with abnormal power, blood-letting is indicated, and the life of the patient may depend upon the prompt abstraction of blood. Notable congestion of the face, a resisting pulse, and absence of hemiplegia, point to a congestion as the pathological condition probably existing. If the patient be young, or below middle age, the probability of this condition, rather than extravasation, is strengthened. *On the other hand, if the apoplexy be dependent upon extravasation, the propriety of blood-letting is doubtful.* It is contra-indicated by feebleness of constitution, advanced age, and if the completeness of the coma denote a large extravasation, or if its situation at the base of the brain be denoted by notable disturbance of respiration and deglutition. It will tend to destroy life if the face be pallid, the surface cold, and the pulse feeble. With the symptoms last named, stimulants are indicated. A large extravasation involves loss of blood, and the substance of the brain is anæmic from pressure of the extravasated blood. Blood-letting will neither remove nor diminish a clot. If the extravasation be not sufficiently large, nor so situated as to occasion a fatal attack of apoplexy, the abstraction of blood may stand in the way of recovery from the shock, and the prolongation of life until the clot is absorbed. *There is no reason to believe that blood-letting tends to prevent or diminish the cerebral hemorrhage.* Cruveilhier, Rostan, and Aussaguel have each reported a case in which hemiplegia ensued directly after blood-letting. I have met with a case in which an attack of apoplexy with hemiplegia immediately followed a profuse hemorrhage from the rectum. Here, as in other

instances, blood-letting is neither to be interdicted nor enjoined in all cases. It will be useful or pernicious, according to the discrimination with which it is employed. *In view, however, of the fact that apoplexy generally involves extravasation, the measure is clearly indicated in only a small proportion of cases.*"

Trousseau, in his "Medical Clinic," asserts, "I not only abstain from all energetic treatment when the symptoms of cerebral hemorrhage are slight, but I even refrain from doing so in very grave cases; in fact, in all cases of apoplexy." The following is a summary of his reasons for such practice:

1. Experience taught him that patients do better without blood-letting, purgatives, or revulsives. The hemorrhage is "*un fait accompli*" at the time when our active co-operation is generally sought, hence our inability to render any service. He asks what influence the above remedies can exercise upon the clot, which is a foreign body present in the brain.

2. He entertains the opinion that "furthermore, far from being useful, blood-letting has seemed to be hurtful, and I believe that *it favors, instead of preventing, congestion.*"

He even expresses the belief that apoplectic phenomena are in some measure more allied to syncope than to congestion, and that bleeding is therefore contra-indicated, not demanded.

This view somewhat coincides with the views recently advanced by Niemeyer, who maintains that the apoplectic phenomena do not depend upon *pressure*, but that the paralysis of the functions of the brain is attributable to "sudden compression of the capillaries, producing *anæmia* of the brain-substance." A large clot necessarily exercises such capillary compression, and prevents the conveyance of arterialized blood to innumerable vessels upon which many nerve-filaments and ganglion-cells depend for their normal supply of nutritive juices. In fact, Niemeyer rejects entirely the old theory that very small clots and extravasations from capillary ruptures can give rise to apoplectic phenomena, "because the escape of blood from the ruptured capillaries can only last till the tension of the contents of the skull equals that of the blood in the capillaries." In accordance with this, the apoplectic phenomena do not follow capillary rupture, owing to the sparseness of the extravasation, and the inconceivably limited anæmia following; the capillary tension is so slight that the tension of the contents of the skull more

than suffices to overcome the mischief, and rapidly places a limit to its results. It is different, however, when an artery gives way, because here the tension required to limit the extravasation is much greater than the capillaries can resist, hence capillary anæmia immediately ensues, the results of which last until the establishment of a collateral hyperæmia more than compensates for the obstruction of the capillaries around the seat of extravasation by which they are rendered impermeable for arterial blood, thus entailing the apoplectic phenomena or the paralysis of brain-function. Hence we can understand that static pressure from the clot does not play so important a part as capillary anæmia in the genesis of apoplectic phenomena, and that Trousseau was not far from right in contending that the latter were more intimately associated with the pathology of syncope, in so far as the existence of anæmia is concerned, than with the effects of congestion.

Dr. Todd, in his remarks upon bleeding in apoplexy, says,—

“There is a practice, unfortunately too common, but which I think is every day becoming less common, namely, that of following an attack of apoplexy by depletive measures, very much as a matter of course. However applicable such a mode of treatment may be to strong, young, hale, and plethoric subjects, I presume no one will say that it is very well adapted to patients who have passed the meridian of life, whose blood and tissues are more or less contaminated by morbid matters, and with whom a morbid state of the arteries of the brain has already greatly weakened the nutrition of that organ.

“With reference to this question of depletion in apoplexy, I would refer to an interesting and very useful work by Mr. Cope-man, in which he has collected, from a great variety of sources, a large number of cases which presented the symptoms of apoplexy. Of 155 cases in which the treatment was specified, 129 were bled, and only 26 were not. Of the 129 who were bled, 51 recovered, and 78 died,—the recoveries being about 1 in $2\frac{1}{2}$, the deaths 1 in $1\frac{2}{3}$. Of the 26 who were not bled, 18 survived and 8 died,—the proportion of recoveries being 1 in $1\frac{1}{2}$; of deaths, 1 in $3\frac{1}{4}$. Eighty-five of the cases were bled generally and copiously, and of these only 28 recovered and 57 died;—in other words, two in every three cases terminated fatally. I am quite aware that the small number of cases not bled casts some doubt on the validity

of the conclusion to be drawn from the comparison of the results of the treatment; but the fact that considerably more than half of those treated by bleeding died (and we owe much to the industry of Mr. Copeman for bringing it out) is a highly significant one, and should arrest attention."

After alluding to cases in which bleeding is productive of successful results, and guarding the practitioner against the adoption of this method in other cases in which it is contra-indicated, he concludes his remarks upon this subject with the following résumé:

"On the whole, then, I think that the results of experience denote that the majority of cases of apoplexy are best treated by purging, shaving the head, and keeping it cool,—perhaps blistering,—and that bleeding is rarely applicable, except to the young, vigorous, strong and plethoric."

Niemeyer, one of the ablest exponents of the modern German school, gives his views upon the treatment of cerebral hemorrhage as follows:

"If cerebral hemorrhage has occurred, it becomes our object to prevent a continuance of the bleeding, to induce re-absorption of the extravasation, and the formation of an apoplectic cicatrix. *But we must not deceive ourselves as to our power, and must understand that we have no remedy for arresting hemorrhage, or for hastening the re-absorption and cicatrization.* In the treatment of this disease, we are restricted to combating the more dangerous symptoms as well as possible. Not a few patients, in apoplectic fits, recover consciousness during venesection, and it seems as if we could, not unfrequently, prevent the extension of the paralysis from the cerebrum to the medulla oblongata, which is indispensable to life, and so save the patient by bleeding. On the other hand, there is no doubt that, in many cases, bleeding during an apoplectic fit hastens a fatal result; collapse occurs immediately after the venesection, and the patient never arouses. . . ."

The indications for bleeding, he considers, can be very exactly given:

"In order that *as much arterial blood as possible may enter the brain*, we must try to facilitate the escape of the venous blood (*venous congestion favoring the anæmia*), without, however, diminishing the propelling power too much. If the impulse of the

heart be strong, and its sounds large, if the pulse be regular and no signs of commencing œdema of the lungs exist, we should bleed without delay. Local bleeding, by leeches behind the ears, or to the temples, or by cups to the back of the neck, cannot replace general bleeding, but may be used as an adjuvant. If, on the contrary, the heart's impulse is weak, the pulse irregular, and rattling in the trachea has already begun, we may be almost certain that bleeding would only do harm,—since the action of the heart, which is already weakened, would be still more impaired, and the amount of arterial blood going to the brain would thus be still more decreased."

From the authorities quoted and the weight of evidence accumulated upon this subject, it is clear that venesection, so long the sheet-anchor in the treatment of cerebral hemorrhage, is a measure of very doubtful propriety, and is to be decided upon only after a most careful scrutiny and exhaustive analysis of the features of each individual case, and of the indications which would seemingly call for its performance. That it has long constituted the measure upon which the principal reliance has been placed in these cases, no one acquainted with the literature of this subject can doubt. That it is the error into which routinists and the faithful followers of staid systematic writers upon medical science are in constant danger of falling cannot be questioned. Yet few who realize the perplexities and intricacies of the diagnostic and pathological bearings of the question will be disposed to deny the great danger of bold and indiscriminate bleeding in these cases. If I have succeeded in inspiring you with the necessary prudence in deciding upon the treatment applicable to cerebral hemorrhage, and have convinced you that venesection is not the *sine qua non* which is immediately to be resorted to when a diagnosis of cerebral hemorrhage has been made, I am more than satisfied, as it is not my desire to indoctrinate you with the views and theories of any particular school, but to place at your command a collection of the necessary facts from which, after patient and earnest reflection, you must form your own conclusions and upon which you should rest your conscientious convictions. It will not avail any man to base an apology for any particular line of treatment upon the argument that if he sins he does so in good company, having the weight, sanction, and authority

of the vast majority of the profession in his favor. Our consciences can alone constitute the stronghold in which our reasons for action are wellnigh impregnable, provided we manfully act only *when we have a right to do so*,—that is, after a most careful investigation of the question, when, with our opinions matured and unbiased, we act according to the best lights we possess. Assuming, then, that our doubts are resolved as to the vexed question of treatment in these emergencies, we must not rest upon the fruits of the satisfaction which never fails to accompany the triumph achieved by sincere efforts to overcome difficulties and reach a substantial ground upon which personal opinions and scientific convictions may be founded. We must be sure not to be led astray in our diagnosis, upon which a safe prognosis and judicious treatment can alone be ventured. By the deductive method, in doubtful cases, we must reason, until we can diagnosticate by exclusion the pathological interpretation of the apoplectic phenomena. Every organ must be interrogated, every symptom analyzed, the history of the case carefully scrutinized, before we can obtain reliable data upon which to rest. Cardiac, pulmonary, or renal diseases, the age of the patient, the gouty, syphilitic, or rheumatic diathesis, the condition of the muscles and of the peripheral arteries, the persistency of the symptoms, the family history, are of great significance, and constitute reliable information, without which no diagnosis can rest with safety. Cardiac and pulmonary lesions, or the previous existence of endocarditis, would strongly point towards emboli in the cerebral arteries, especially in the left arteria fossæ Sylvii, which seems to be one of the principal seats of obstruction,—almost by selection, we might venture to say, in ignorance of any anatomical explanation of this common occurrence, which should never escape our attention in post-mortem investigations. The age of the patient is essentially important, knowing, as we do, that in persons advanced in years atheromatous degeneration of the arterial coats is the common cause of thrombosis,—a condition so apt to be attended with symptoms of chronic brain-disease in old persons affected with atrophic softening. The poisons of syphilis, rheumatism, and gout play an important part in these cases by the tissue-poisoning produced: the two latter especially, by the irritation they occasion in the urinary organs, and the improper elimination, through that channel, of excrementitious substances,

become the cause of brain-disease by qualitative blood-changes, from which source the nervous tissues suffer the results of faulty and improper nutrition.

The condition of the peripheral arteries is of importance only in the clue it furnishes us to the condition of the cerebral vessels, upon which, however, for reasons previously assigned, too much reliance is not to be placed. The persistence or variation of the symptoms is of great assistance to us, for the following reasons. Certain authorities maintain that changes in symptoms in chronic cerebral disease, the appearance and disappearance from time to time of paralysis, are almost pathognomonic of softening. This fact must be admitted with a certain allowance, as such phenomena are explained by the re-establishment of the collateral circulation; but when a large, important artery is plugged, the symptoms no longer appear and disappear, as the functional disturbances will then necessarily remain permanent. The seat of thrombosis and embolism is also to be taken into consideration, as the collateral circulation is more readily or less easily affected according as the obstruction of the circulation is beyond or below the "circle of Willis."

The state of the muscles; according to Dr. Todd, is a matter of no trivial significance in cases in which paralysis follows apoplexy, both as to their apparent condition and their electric contractility. His views may be briefly enumerated as follows: 1. Muscles *relaxed* completely immediately after an apoplectic seizure, point to an atrophic state of the brain. 2. Muscles contracted *immediately* after the fit, correspond with a state of brain-irritation. 3. Muscular rigidity occurring *late*, is indicative of an attempt at cicatrization: this spastic condition of the muscles acquires additional weight if associated with muscular atrophy. In muscles affected with early rigidity, the galvanic excitability of the unsound limb is greatly increased, and in cases where complete relaxation is manifested, the galvanic excitability of the paralytic limb is much less than that of the one on the opposite side not paralyzed.

Now, gentlemen, I shall conclude with a few remarks on the proper treatment in cerebral hemorrhage. From the evidence of statistics quoted, and the views of the eminent authorities cited, strengthened as they are with weighty arguments; from the accu-

mulated strength of our own convictions ; from the fact that when we are called upon for help the damage is already accomplished ; from our inability, having no available therapeutics, to remove a clot from the brain, or to remedy the evils of the laceration, destruction, and disorganization it has effected whilst ploughing through the delicate cerebral structures, in the midst of the bloody wreck of the neuroglia, ganglion-cells, and nerve-filaments,—we are compelled to admit our helplessness, and, realizing the hopelessness of benefit accruing from our efforts to ameliorate a destruction already achieved, we must desist from all measures that might aggravate the critical condition in which we find the unfortunate victim of cerebral hemorrhage. But are we to remain passive witnesses and with folded arms await our patient's death ? By no means. Whilst we must not over-estimate our ability, we are justifiable in essaying what an enlightened pathological knowledge holds forth as inducements of success. We must obviate the tendency to death,—so far as we may be able,—and we must battle with those complications and accidents which may arise. In some cases we may bleed “in spite” of the apoplexy,—that is, admitting that in given cases we may have conditions of depression co-existing with those of irritation, as illustrated in an atrophic brain, where the establishment of the collateral circulation becomes excessive, producing the symptoms of partial hyperæmia, the so-called “*Herdsymptome*” of Griesinger, thereby causing danger of a recurrence of the extravasation, or productive of dangerous œdema. In such cases a moderate venesection, through the mechanical relief it affords, might be the means of *saving life*. This corresponds with Niemeyer's advice, to bleed in certain cases of pneumonia, “in spite of the pneumonia,” which pneumonia is a contra-indication for bleeding. He cites cases in which the collateral hyperæmia in healthy portions of the organ becomes a source of such danger through consecutive œdematous infiltration that not to bleed would be to sacrifice the patient. Here the author clearly means to bleed in spite of the disadvantages accruing from loss of blood in inflammatory diseases, in order to obtain mechanical relief by the diminished arterial tension produced in the hyperæmic portions of the lung endangered by too violent fluxionary hyperæmia. Admitting, therefore, the possibility of a *primary* atrophic condition of *depression* co-existing with a *second-*

ary hyperæmic state of *irritation* in the same brain, based upon well-understood and universally admitted pathological laws, we would unhesitatingly bleed in *some cases*, carefully to be selected and discriminated, and in which we would not dread the effects of the additional shock of venesection. This we the more readily admit in consequence of the danger to be anticipated from too violent an establishment of collateral fluxion, which, by its pressure, when accompanied with excessive resulting œdema, the evil effects of which are sometimes conveyed to the healthy hemisphere in spite of the protection of the falx cerebri, intensifies the patient's danger and diminishes his chances of recovery. The anatomical arrangement of the falx has been considered of much importance by Niemeyer, who lays stress upon the fact of its dipping deep between the *posterior* portions of the hemispheres, thereby affording greater immunity from pressure exercised in that situation than is the case when tumors or extravasations are situated in one of the anterior lobes, where the attachment of the falx to the crista galli furnishes more facility for the transmission of the consequences of such disturbing causes from one anterior lobe to the other.

In the vast majority of cases, therefore, we would not bleed; and here we will divide our patients into two classes, requiring totally different modes of treatment. In a patient who is affected with complete hemiplegia, suddenly, without loss of consciousness, and in whom, therefore, we have no difficulty in differentiating white softening from sanguineous extravasation, there is little to do beyond placing him in a horizontal position, avoiding all sources of excitement, and giving him the benefit of the best hygienic measures, and perhaps a brisk purge. In the second class of cases, those suddenly rendered completely hemiplegic, with loss of consciousness,—provided the pulse is strong, full, firm, and slow, the heart healthy, and there are evidences of great congestion, as evinced by flushed face, turgid skin, and injected conjunctiva,—we may undoubtedly take a moderate quantity of blood by cups applied to the occiput, and in some instances, should the symptoms of irritation become very threatening, may resort to a moderate venesection. In case the patient has been seized with a fit during a long and luxurious meal, knowing how a distended stomach may mechanically provoke excessive cerebral hyperæmia,

we would be justifiable in administering a stimulant emetic, in the selection of which remedy we must aim at combining rapidity of effect with the avoidance of prostration. In cases in which a *medium* course is to be pursued, the head may be shaved, cold cautiously applied, and the bowels freely moved, avoiding, of course, excessive purgation. In cases in which the patient is rapidly sinking, death by syncope impending, sinapisms, stimulants, and restoratives are to be energetically used. Such cases, it is needless to add, you will recognize by the rapid, feeble, irregular and intermittent pulse, the pallid countenance, and the cold skin. During the stage of so-called reaction, when the dangers of encephalitis are to be apprehended from the presence of the clot, we must energetically try the effects of cold to the head and of warmth to the lower extremities, and perhaps the internal administration of salines and of the iodide and bromide of potassium. Having no confidence in the beneficial influence of mercurials to produce the absorption of the clots, and believing that they would only be potent to produce a cachexia that would thwart the *vis medicatrix naturæ* upon which we must depend for the absorption, disintegration, and encysting of the clot, we must entirely eschew them in our practice. As the violence of this stage subsides, blisters, pustulation, or other counter-irritants might, by their reflex influence, be of service. In the treatment of the hemiplegia we strictly avoid the administration of strychnia, accepting as we do the opinion of Dr. Todd, that a sort of elective affinity for the strychnia is exercised by the portions of the nervous substance affected, and that the most baneful consequences may result, without any compensating advantages. The benefits of faradization, properly employed and not used too early, cannot be questioned, and it forms one of the resources upon which the most reliance can be placed for treatment of the hemiplegic symptoms. Country air, mild diet, and the strict observance of hygienic laws are of essential importance.

Before concluding, we have yet one recommendation to offer in reference to our timely interference in certain cases, and the *prevention* of cerebral hemorrhage, which, once *un fait accompli*, makes our inability to render much service to our patients oftentimes so sadly evident. Unfortunately, the physician is sent for too late, and his hands are tied. This is, however, not the case

when, in the course of practice, cerebral hyperæmia attracts our attention, and should excite our earnest endeavors to avert the threatening cerebral hemorrhage. Here is a critical moment for the patient, as upon the skill and decision of his physician depends his life, or at least his rescue from imminent danger. It is in such instances that the votaries of the healing art feel inspired with enthusiasm and admiration for the science of medicine. By an acquaintance on the part of the physician with the laws and mode of causation of fluxionary and passive cerebral hyperæmia, the patient will be saved, as if by magic, from destructive lesions. We deduce from the statements made that, first, in all cases of *active* or *fluxionary* hyperæmia of the brain, whether occasioned by undue increase of cardiac action, obstructed arterial escape of the blood from the aorta into other branches, or vaso-motor paralysis; and, in the second place, in all cases of *passive* or *congestive* hyperæmia, whether superinduced by compression of the vena cava descendens and jugular veins, by energetic expiratory movements while the glottis is contracted, by cardiac affections with functional disturbances without compensating muscular hypertrophies, or by compression of vessels in, and disturbances of, the pulmonary circulation,—unless the treatment be prompt and energetic, cerebral hemorrhage is greatly to be dreaded. To prevent this, the measures best calculated to remove the hyperæmia are at once to be resorted to; and in many of the instances referred to, the mechanical relief afforded by a judicious and well-timed venesection is as astonishing as it is beneficial. Various other appropriate remedies, which it is not now necessary to enumerate, will aid us in our endeavors to avert the evils of cerebral extravasation, which, once it has occurred, in many cases makes the physician's helplessness as painful as it is mortifying.

LECTURE XVII.

ENCEPHALITIS.

Definition.—Causes: Injuries, Cerebral Hemorrhage, Neoplasms, Caries of Cranial Bones, Infectious Diseases.—Red Softening.—Forms of Termination.—Diagnosis.—Symptoms.—Hemiplegia.—Prognosis.—Treatment.

GENTLEMEN,—I propose to-night to speak of encephalitis. By this is meant an inflammation of the substance of the brain. If you recollect the repeated allusions made to the anatomical composition of this organ, you are aware that it has little connective tissue, or neuroglia, which latter term was invented by Virchow, its literal meaning being “nerve-glue,” as applied to the ordinary connective tissue. It is its paucity which gives certain marked peculiarities and characteristics to inflammation of the substance of the brain. Encephalitis is an inflammation of the cerebral pulp. It is a rare form of affection: still, you are liable to meet with it, and the very first case in your practice might be of this character, and hence the necessity of your being able to recognize it. In encephalitis an inflammation occurs in the neuroglia, the nerve-fibres, and the ganglionic cells, the ultimate constituents of the nervous substance.

The first thing we witness in inflammation of the brain which is not exudative in character is a “cloudy swelling,” a filling up by increased nutrition of the cells, whose contents are proportionately increased. This is what the French call *engonflement*, an increase in the nutritive juices of the brain-tissue proper: hence the nerve-cells are swollen in appearance, and become cloudy. After this condition has existed for a certain length of time, certain retrograde processes, as fatty degeneration, occur, and as a result detritus is formed in the inflamed parts in the form of a milky deposit, consisting of debris of nerve-filaments and nerve-corpuscles of the brain. In giving you this description I have explained the primary pathological states of the inflammation. I

shall revert to the description of their subsequent developments when I come to speak of the anatomical appearances.

Although a rare disease, encephalitis may occur in a very limited practice. Perhaps you recollect that in my lectures upon meningitis I have often said that where ordinary (acute idiopathic) or basilar meningitis exists, there is constantly an inflammation of the pia mater and arachnoid membranes, and that often in attempting to detach the former from the pulp of the brain, in autopsies, it happens that the membranes are not easily separated, from the fact of the inflammation having extended to the peripheral portions, and consequently there is an adherence of the pia mater to the hemisphere, causing the pulp to be torn upon an attempt at removal of the membrane. You will, perhaps, ask me if this be not a true encephalitis,—a meningitis *plus* an encephalitis. Of course, to all intents and purposes we have here two forms of inflammation co-existing; but we have not the particular form implied by the term encephalitis. In this disease there is no involvement of the membranes, as it is only an inflammation of the pulp, without anything of a meningeal character.

What conditions and etiological factors lead to encephalitis? In the first place, traumatic injuries, whether of the coverings of the cranium or of the cranium itself. All injuries or blows to the head may thus result: and this is really an interesting point. Any such blow, even though originally of trivial importance, may at some future time be the superinducing cause of a severe encephalitis. Forbes Winslow states that apparently slight blows have been the source of disastrous effects, occurring sometimes many years after the original injury. This is the reason why blows upon the head should always cause serious apprehensions and be a source of anxious solicitude. Encephalitis is an insidious disease, in which therapeutic measures are not of much avail.

But how do concussions produce encephalitis? When a man receives a serious blow upon the head, there may be an extravasation of blood, or a depression of bone, or concussion of the brain-fibres. You all understand that, if the blow be never so slight, a rupture of the capillaries may occur, attended by a minute extravasation of blood, even so minute as to escape observation in an autopsy, supposing an opportunity to have been accidentally

furnished. The foreign body—be it only a few drops of blood—will act as an irritant, and, *ubi irritatio ibi affluxus*, a slow but progressive inflammatory condition leading to encephalitis will ensue.

Another cause of this disease is cerebral hemorrhage. When lecturing upon that subject, I told you that if the patient survived he generally remained hemiplegic, and it often happened that in a few days symptoms of irritation were developed, consisting of fever, headache, hyperæsthesia, with increased temperature and vascular action, which pointed to the fact that encephalitis had been excited by the clot. Cerebral hemorrhage should therefore be placed under the head of traumatic causes.

Another cause is neoplasms. This includes all the different forms of adventitious growths, such as scrofulous, cancerous, and syphilitic tumors, etc. The foreign bodies encroaching upon the brain become a source of irritation and finally of inflammation.

The next cause brings back to mind pachy-meningitis. You remember that the most frequent origin of pachy-meningitis is caries of the cranial bones, due to otitis internà, ozæna, etc. The same is not infrequently a factor of encephalitis. A long-continued otorrhœa, ozæna, etc., by inducing disease of the cranial bones, and idiopathic, traumatic, or syphilitic affections of the osseous structures, will lead likewise to encephalitis by a propagation of the inflammation to the substance of the brain. If you have a patient with a long-standing ozæna and presenting brain-symptoms, always dread the probable existence of pachy-meningitis or encephalitis. Though you may not be able to arrest the disease, having made a proper diagnosis you can furnish the patient's friends with satisfactory information, which will be as pleasing to them as to yourself.

It is unnecessary for me to discourse minutely upon the symptoms and burden your memory, considering that we have so few satisfactory data for the therapeutic management of such cases. But, as I have said, it is always important to diagnosticate correctly, even where we are unable to effect a cure. In reference to the subject under consideration, you will all readily appreciate the importance of disease of the cranial bones.

The next cause of encephalitis embraces all the different forms of infectious diseases, such as glanders, pyæmia, etc.

In regard to the anatomical appearances in this disease, remember that it is almost invariably a circumscribed affection, being rarely diffused. It occurs mostly in patches, perhaps the size of a pea, a hazel-nut, a hen's egg, or your fist. It may be deep-seated or superficial, cerebral or cerebellar, is generally situated in the gray portions, and especially affects the superficial parts, the peripheral portions of the convolutions of the hemispheres. In this disorder the tissue-destruction occupies circumscribed limits, or the inflammatory patches are reduced to a peculiar reddish pulp, having a rusty appearance, and of a color not unlike that which is characteristic of the sputum in pneumonia. When examined with the microscope, it is found to consist of the detritus of ganglionic cells, nerve-filaments, and neuroglia, with an accompanying mass of fine granular exudations.

The inflammation may assume three different forms. First, in the circumscribed spots a proliferation of connective tissue occurs; the watery portions are absorbed, and the solid parts are rendered firm; finally they shrink, and the cavities thus left are sometimes filled with a milky fluid. Secondly, several small cysts on the peripheral parts of the hemispheres coalesce, afterwards shrink, and constitute the cicatrices which are evident upon post-mortem examinations. The third, or common termination, is the formation of pus, resulting in cerebral abscess,—a termination much to be dreaded. In fact, nearly all abscesses of the brain originate in circumscribed encephalitis, which becomes encapsulated with pyogenic membranes. The abscess, of course, occurs wherever the encephalitis was located, which is generally near the periphery of the hemispheres. Often it extends deeply into the substance of the brain; and in proportion to the encroachment upon important parts will be the severity of the symptoms.

An important fact, which I wish to impress upon you, is that this affection, like cerebral abscesses and tumors, presents no pathognomonic symptoms. The result is, that an abscess has often been mistaken for a tumor, and *vice versa*. No one can diagnose either by the symptoms alone. The diagnosis can be arrived at only by following the rule laid down in the beginning of this course in regard to other affections, which requires the etiology of the case to be studied, the history to be thoroughly

elucidated, and the sequence of events to be duly considered. Would you think of diagnosing typhoid fever or meningitis by one symptom? Were you to attempt it, you would constantly err. There is scarcely a pathognomonic symptom of any disease. It is by grouping many of the more important features of a malady that you are enabled to effect its recognition. From my previous remarks you know that abscesses in the brain, or even encephalitis, may exist without their presence being suspected during life; that is, if situated in those parts which do not essentially contribute towards the performance of vital functions. It is stated by Trousseau and others that large tracts of brain-mass are often softened from inflammation without the manifestation of symptoms during life. This is accounted for by the fact that the brain is a dual organ, so that when one part is diseased or unable to perform certain functions the other compensates by doing double duty, as is the case with the lungs and other dual organs. But if the part affected be in the tubercula quadrigemina, pons varolii, medulla oblongata, or mesocephalon, or if there be an encroachment upon other vital parts, serious symptoms will be the result, and they will be urgent in proportion to the importance of the parts affected.

The symptoms of abscess of the brain and of encephalitis are of the following character. First, they depend upon the portion of the brain involved; secondly, upon the disturbances of circulation,—which I more particularly described while lecturing upon hyperæmia of the brain; and, in fact, cerebral affections of every kind produce certain symptoms depending upon and varying according to changes in the circulation. Thirdly, the symptoms depend upon the amount of intracranial encroachment on the districts of the brain implicated. Some of the symptoms apply to encephalitis, and properly depend upon it. Then we have others caused by disorganization of the brain-substance itself. The ordinary symptoms of this affection are limited forms of paralysis, especially if it exist in certain relational centres presiding over the motor nerves. If the disease be in those portions which preside over sensation or the special senses, nervous impressions derived therefrom will be impaired or no longer be appreciated. Whatever functions the affected parts preside over will be involved in the symptomatic manifestations.

As in cerebral abscess, we generally have hemiplegia, the paralysis being unilateral, seldom or never bilateral, unless the affected portion be situated in the median line and below the division of the hemispheres, thus being common to both parts, an occurrence which is exceedingly rare. The abscess or encephalitis being generally above the decussation of fibres, the hemiplegia is generally in the opposite side of the body. In tumors there is less hemiplegia, and usually a more limited paralysis of certain cranial nerves. Although the disorder is insidious, there is generally a sudden outburst accompanied by symptoms of inflammation, announcing the disease. Let us construct our chain of pathological evidence. Suppose, in the first place, a man has received a severe blow upon the head; this will constitute the first link in the chain. At some subsequent period of his history he shows symptoms of cerebral irritation, fever, a quick (though often slow) pulse, headache, hyperæsthesia of the nerves of special sense, dizziness, photophobia, vomiting, etc., with an implication of the nerves of motion or sensation; forming the second link, and confirming our suspicions. If the affection does not rapidly progress, a *lull* now occurs, the interval being often protracted, with a complete cessation of all alarming symptoms: this is characteristic of the disease, and is well calculated to deceive you if you have not studied its natural progress. After the storm comes a bright and seductive calm, often causing the physician to discharge his patient as convalescent, and to be elated at his success in curing an inflammation of the brain. The pause has merely misled him, and in the perusal of the annals of medicine you will find just such cases recorded, in which, upon the subsidence of the symptoms of irritation, the doctor has congratulated himself upon his victory. Watch this patient, however, for a month, a year, many years, and in all probability you will find that, if not carried away by an intercurrent disease, he will die with symptoms of abscess in the brain. I say in all probability, for it is not necessarily so; but generally the death will be the result, clearly, of the antecedent attack of this affection, and will be directly traceable to it, even after the lapse of a great length of time. This will surely teach you that in cases of injuries to the head you are always to be cautious in your prognosis, entertaining well-grounded apprehensions which will cause you to inform both the patient and his friends of the possibility

of future danger. Under these circumstances, in criminal cases, where insanity is suspected, and a previous injury to the head has been proven, you should always give the defendant the benefit of the doubt.

I recollect one poor fellow who would have been sacrificed by the law had it not been for the action of the governor in commuting the sentence of death to imprisonment. He had, while a child, fallen from a barn, so injuring his head that the scars were still visible, and a depression of the cranial bones was still very apparent. Subsequent to the accident epileptic attacks were developed, as was sworn to by physicians in Germany, where this young man was born. Years afterwards, here in St. Louis, he was seized with a fit of epileptic fury and killed his uncle; and yet the jury could see no grounds of extenuation in the facts, and the prosecutors condemned the so-called "maudlin sentimentality" of medical experts. Justice was not asked to forgive a crime, but to give a man the benefit of a doubt, and place him in an insane asylum, that haven of medical protection. This poor fellow has to pass the remainder of his days in company with the worst of criminals, simply on account of his misfortune. This, surely, ought to inspire you with regret; and if my efforts shall have been successful in saving the life of some poor, irresponsible individual, I shall feel amply repaid for my endeavors, and this whole hour will have been consumed to your advantage, even if you have learned nothing else.

After the symptoms of irritation, as I have already told you, the deceptive calm occurs. Do not for a moment imagine your patient to be safe. When abscesses, the natural result of encephalitis, have formed in the brain, one of the prominent characteristics is epileptiform convulsion, and, after pus has collected, rigors, with paralysis, soon follow. Considering the fact that there is no pathognomonic symptom of the disease, you will be more than ever convinced of the inutility of an attempt to give undue importance to the symptomatology. The history and sequence of events should never be overlooked. The duration of the calm may be for a week, a month, a year, or longer: indeed, I recollect the case of a gentleman who became insane fifteen years after having received a blow upon the frontal region. I have already said that the pathological progress is apt to be slow and insidious, and that,

should no important part of the brain be involved, it may not at all interfere with life.

The prognosis is very unsatisfactory, for only a small minority do well, and the large majority die.

I refer to treatment merely as a matter of form. Having ascertained the true nature of the attack, treat it as you would treat any other active brain-trouble, but with due precautions. Should an injury to the head occur in a child at school, forbid all study, place it in a dark room, and make cold applications to the head. Derivatives are good; and active, not drastic, cathartics may be recommended as most efficacious. Keep the head cool, and freely administer bromide of potassium and ergot, to regulate the determination of blood to the brain.

Above all, keep the patient quiet and in a dark room. There is much virtue in this advice,—more, perhaps, than in many remedies “of which we know little, and placed in a body of which we know less.” Resort to local bleeding, and apply cups over the mastoid process of the temporal bone. In short, try to regulate, and, if possible, prevent, the intensity of the storm which you may palliate but cannot arrest.

LECTURE XVIII.

PARALYSIS.

GENTLEMEN,—I shall commence this topic by a careful study of some of the principal clinical aspects of paralysis. This is a subject replete with interest, and of the utmost importance to the practicing physician. The word paralysis means a loss of muscular contractility, and necessarily implies an impairment of the power of motion. Sensation may be more or less affected in these cases: sometimes it is morbidly increased, when the term hyperæsthesia is used; sometimes it is greatly diminished, or even destroyed, when the condition is known as anæsthesia. When, however, we use indefinitely the term paralysis, motion is always understood as being particularly affected, and is therefore the disturbed function to which we particularly allude. Paralysis may be general or only partial. It may affect many muscles, a group of muscles, or only one muscle. When confined to one side of the body, we call it hemiplegia; when it affects the lower half of the body, paraplegia is the term by which it is designated. Again, it may occur suddenly, as we shall hereafter see, or, on the other hand, its invasion may be gradual, insidious, and almost imperceptible. Never lose sight of the fact that paralysis is always a *symptom*, never a *disease*. If you always recollect this, you will invariably seek to make a correct diagnosis of the cause, never contenting yourselves with the mere knowledge of its existence: thus you will apply the proper therapeutic measures to each case, and your prognosis, which is a matter of much importance, will not be founded on error. To illustrate this explanation and impress it thoroughly upon your minds, we will consider what a physician does in cases of dropsy, which, like paralysis, is only a symptom, and never the disease. Is his duty accomplished when he has become satisfied of the existence of dropsy in his patient? Does he not carefully interrogate each organ to ascertain its condition?

Does he not earnestly strive to determine whether his patient is laboring under cardiac, hepatic, or renal disorder, before he makes any attempts at medication? Thus it is in reference to paralysis. Once aware of its cause, and a great part of the problem of prognosis and treatment is solved.

The principal causes of paralysis may be divided as follows. *All lesions of nerve-centres* are apt to be followed by paralysis. Spiculæ of bone, clots of blood, effusions, tumors, inflammations, softening, are all so many causes of paralysis. Any *functional* disturbance of the brain, spinal cord, or great nerve-centres may likewise be followed by paralysis, as, for instance, emotional and diphtheritic paralysis. *Any lesion in the continuity* of a nerve will as surely produce paralysis as will a break or interruption in the course of a telegraph-wire prevent the transmission of the electric force. Any affection or disturbance of the nerves at their *periphery*, or extremities, may result in paralysis; and we have well-marked instances of peripheral paralysis. This variety of palsy is usually local in character. All physicians constantly meet with phenomena due to *reflex nerve-action*, and prominent among them is reflex paralysis. The spinal cord, being the great centre of reflex action, communicates the impressions it receives from one part of the body to distant parts. First it receives the impression, we will say, for instance, from the periphery through the medium of the sentient nerves. The impression is duly recognized in the ganglionic or deep-seated portions of the cord. A new nervous influence is generated, and transmitted almost with the rapidity of lightning to distant parts through the motor nerves, occasioning involuntary muscular action. Hence a diseased or morbidly irritated part may, through the medium of nervous communication or reflex action, influence distant parts of the system. You will therefore perceive that difficult dentition, worms, ingesta, and intestinal irritations may be the cause of fleeting forms of paralysis. A wound of a nerve on one side of the body has been known to occasion paralysis on the other.

Causes which produce *disturbances of the circulation* are sometimes productive of paralysis. Obliteration of important arteries, or plugs of fibrin diminishing or filling up their calibre, may prove very serious causes of palsy.

Paralysis may be due to a *morbid state of the muscles themselves*,

which is a condition totally distinct from the other forms, as then the nerve-influence may be perfectly healthy and normal and yet a decided palsy occur. *Contamination of the blood* with poisons produces many of the well-known clinical aspects of paralysis. Arsenic, lead, mercury, alcohol, the *materies morbi* of gout, rheumatism, diphtheria, etc., operate changes in the economy that interfere with nerve-function. These are, therefore, the principal causes of paralysis; and I shall now occupy your attention with a short description of the best-known varieties, which you will most frequently meet with in practice.

Hemiplegia is paralysis confined to one half of the body, generally affecting all the muscles which are voluntary. The limbs are, as it were, dead, deprived of the powers of motion and sensibility. The cheek on the same side as the paralyzed limbs hangs; the mouth is drawn towards the healthy side, in consequence of a paralysis of the muscles on one side, which no longer antagonize those on the healthy side,—hence the healthy muscles draw it to their side; the tongue is ordinarily pushed out towards the palsied side, because the muscles which protrude the tongue on the healthy side alone act; those on the affected side remaining motionless, the muscular contractions on the opposite side force the tongue towards the side where there is an absence of action, diminished resistance, or cessation of antagonism. Articulation is imperfect, and at times it is quite difficult to interpret the expressions of the unfortunate patient. Brain-lesion is the common cause of hemiplegia. We would therefore naturally expect to find more or less evidence of mental impairment or a disordered condition of the intellectual manifestations; whereas, in spinal lesions producing paraplegia or hemiplegia from an involvement of the upper half of the cord, such symptoms are entirely absent.

Taking it for granted that we have ascertained the hemiplegia to be of cerebral origin, the next important thing that presents itself to our mind is to determine the *character*, and then, as far as possible, the *seat*, of the lesion.

This latter point will not present so many difficulties as the other. It will be sufficient here to state that the lesion is always situated on the side opposite to the paralysis, provided the lesion (which is generally the case) is situated above the point of decussation of the fibres of the medulla oblongata. A lesion on a level

with this decussation would necessarily involve both sets of fibres, and consequently produce a double-sided palsy. A lesion below the point of decussation would be followed by palsy on the same side as the lesion. Now, as the corpus striatum is believed by most physiologists to be connected with the generation of motor impulses, should motion be particularly involved in the paralysis, we would readily conclude that to be the nerve-centre most implicated. On the other hand, as the thalamus opticus is credited with the production of the sensory phenomena, an impairment of sensation would immediately suggest to our minds its disturbance. These suppositions are to a certain extent correct in theory ; but so intimate is the connection of these two bodies that one is rarely involved without a corresponding damage being inflicted upon the other.

The *nature* of the lesion can be determined only after a careful consideration of all the facts of the case. Récamier, Trousseau, Todd, and other authors state that an absolute and complete paralysis coming on *suddenly*, and accompanied with profound coma, is almost pathognomonic of serous or sanguineous apoplectic extravasation. On the other hand, paralysis which, though complete and *sudden*, is unaccompanied by coma or loss of consciousness, is almost certainly caused by white or atrophic softening of the brain. Paralysis occurring *gradually* and insidiously is generally the result of chronic brain-disease, viz., abscesses, tumors, exostoses, red softening, and other brain-affections, which slowly develop themselves and very gradually exercise increased pressure upon the great cerebral centres. The age of the patient must also guide us in our diagnosis, as after a person has passed his fiftieth year atrophic softening frequently manifests itself by complete and unexpected paralysis, without any evidence of the comatose stage.

Our task of unraveling the character of the lesion does not end here. We must explore every organ of the body minutely and carefully, in order to ascertain the cause of the paralyzing lesion. *The heart* must be very carefully auscultated, as any indications of cardiac disease would immediately awaken our suspicions and direct our attention to the circulating organs as the *fons et origo* of the nervous trouble. These suspicions would almost become convictions did the previous history of the case plainly, in connec-

tion with cardiac lesion, establish the existence of the rheumatic diathesis. Cardiac troubles, and especially hypertrophy, lead to apoplectic extravasations into the brain. Or it may happen that a fibrinous concretion is washed into the blood-current from one of the heart's valves, or a small fibrinous clot may be hurried along and finally impacted in a cerebral artery, effectually plugging it, and interfering with the nutrition of the part of the brain thereby supplied, thus eventuating in atrophic softening.

Then, again, the *kidneys* may be at fault, and in consequence of their disease excretion is imperfectly performed, the blood becomes overcharged with excrementitious matters, nutrition is at length impaired, and an atheromatous condition of the arteries is finally developed; all this in consequence of a poisoned blood, which in its turn produces chronic brain-troubles, the last link in this long chain of pathological changes.

We have still other expedients to aid us in determining the character of the lesion, among which are the well-known electrical observations of the late Dr. Todd. This distinguished author divides the condition of the muscles after paralysis into three distinct states. When *muscular contraction* or *rigidity* occurs *early* or immediately after the development of the paralytic symptoms, according to him, the indication is that of an "irritative" brain-lesion, such as apoplectic extravasation, inflammation, or puriform accumulation in the subarachnoid spaces. Should the muscular *rigidity* develop itself *late*, and be accompanied with muscular atrophy, he attributes the above-mentioned phenomenon to "an irritation from cicatrization" occurring in the cerebral tissues. Finally, should *relaxation* be the prominent muscular characteristic, we may conclude that the lesion is owing to some "depressing" cause, such as white softening, with or without rupture of the blood-vessels. The response to the electric stimulus will, in the first instance, be increased, in the latter will be greatly diminished.

By *paraplegia* we mean a paralysis of the lower half of the body. This paralysis is double,—that is, it differs from hemiplegia in affecting both sides of the body; but it invariably affects the lower extremities. The cause of paraplegia is usually of spinal origin, the brain being unaffected. Hence in this form of paralysis we rarely or never have any symptoms of disturbed or

impaired intellection, the spinal cord being the medium by which the brain's mandates are conveyed, and not in any manner presiding over or generating the intellectual phenomena. Functional disturbances of the cord, without any evident lesion, may likewise produce paraplegia. Its invasion is generally slow, and at times difficult to appreciate in its incipency. One side is frequently more affected than the other, and diminished sensibility is almost an invariable accompaniment of the diminished or totally extinct motor power.

To demonstrate the progress which has been made in the study of nervous affections, I shall present for your consideration a form of spinal paralysis which fifty years ago would not have been diagnosticated, and which is certainly understood with extreme difficulty. For this hypothetical case I am indebted to the valuable contributions and lectures of Dr. Brown-Séquard. We will suppose that a patient comes to us with a complete paralysis of motion on one side, which, for convenience, we will imagine is the right, and that on the same side, besides a markedly increased temperature, a permanent and marked hyperæsthesia exists. On the opposite or left side is a total abolition of sensation, with diminished temperature, but perfect motor capacity. This paralysis, we will suppose, is absolute and complete from the neck downwards. The hyperæsthesia, or increased sensibility, deserves a passing remark, as one of the most important recent advances in physiology consists in Dr. Brown-Séquard's demonstration of the fact "that nerve-conductors of the various kinds of sensitive impressions and of the reflex phenomena, and also those for the transmission of nervous force to muscles, blood-vessels, etc., are absolutely distinct one from the other as regards their functions. For instance, there are four distinct kinds of nerve-fibres of the higher senses. *Touch*, or the faculty of sensation, causes the appreciation of the object brought in contact, and its qualities, properties, and characters. The next nerve-fibre we have to consider as being a conductor of one of the four kinds of the higher senses is the one which conveys sensations of *tickling*, which is a sensibility quite distinct from the other. There is also a fibre capable especially of transmitting *painful* impressions, such as a pinch or prick. Lastly, a fibre the appropriate function of which is that of conveying to the brain due appreciation of *temperature*,

or cold and heat. Hence we have distinct varieties of sensation, each having appropriate conductors, *totally distinct* and perfectly independent one from the other as regards their function." Our patient is affected, therefore, with a complete paralysis of one-half of the body, from the head downwards. On the same side of the body on which paralysis of motion exists he has increased sensibility, manifesting itself by extreme sensibility to touch, tickling, pain, and temperature; and in addition to all this, as we have already stated, the temperature is increased. On the left side, of course, we will have an anæsthesia of these four kinds of sensibility. In the *face*, on the side of the injury, there are an increased heat, an increased sensibility, and a contraction of the pupil. Now, how shall we explain the lesion which produces these symptoms? The pathological condition which we should expect to find would be an injury of the entire lateral half of the spinal cord. The paralysis of motion is on the right, or side of the injury, because the motor nerve-fibres "emerge from each side of the spinal column in such a manner that the fibres which control the movements of limbs on the right side pass to the right side;" whereas, on the other hand, "the sensitive nerve-fibres, which serve to the first four kinds of sensibilities we spoke of, pass into the spinal column in such a manner as to go to the other side of that organ, so that the nerve-fibres of sensibility in my right arm and right leg pass into the *left* side of the spinal column." Hence impaired sensibility exists on the side of the body opposite to that of the injury. It may be well here to remark that the sensitive nerves decussate through the entire length of the spinal cord, and that the motor nerves decussate only in the anterior pyramidal columns of the medulla oblongata. Late physiological researches seem to establish the fact quite clearly that the posterior columns are more intimately associated with sensation and the co-ordination of muscular movement, while the anterior columns are connected with motor phenomena. Hence in some diseases, as in progressive locomotor ataxia, where muscular co-ordination is particularly affected, the posterior columns are found seriously disorganized. The increase of heat in the right side is readily explained by the involvement of the sympathetic on the same side of the injury, as any lesion occasioning paralysis of this nerve necessarily affects the vaso-motor nerves which are derived

from it, and we then must necessarily have the well-known congestions and increased temperature so ably demonstrated and first alluded to by Marshall Hall when he experimented by dividing the cervical sympathetic. This hyperæmia has some analogy to inflammation, but is at the same time convincing proof, as Virchow claims, that hyperæmias may and do exist without necessarily being followed by inflammatory changes, because suppuration, ulceration, gangrene, œdema, etc., never follow the division of the sympathetic, although the resulting congestions sometimes last weeks and months.

In paralysis, the nervous influence is not always at fault, and we must sometimes strive to ascertain whether the muscles themselves are not the seat of disease. A well-marked form of motor deficiency is found in an affection known as *progressive muscular atrophy*,—progressive, because of the persistent and oftentimes rapid progress made when once the symptoms are well established; muscular, from the fact that the pathological changes occur in the ultimate muscular fibres themselves; atrophy, because of the characteristic wasting of the muscles. The recent advances in microscopic investigations prove beyond any probability of mistake that fatty transformations manifest themselves in the muscle-cells and occasion the morbid changes and consequences which inevitably follow upon such errors of nutrition. The pathognomonic symptom “is a constantly increasing inability to perform certain muscular movements.” Muscles thus affected dwindle, are prone to peculiar twitching movements, and present a peculiar soft and yielding sensation to pressure. The muscles of the hand, the flexors and supinators of the fore-arm, the biceps, and the deltoid, are most likely to be involved.

LECTURE XIX.

EPILEPSY.

Leading Symptoms.—Abolition of Consciousness pathognomonic.—Premonitory Signs.—Aura Epileptica.—Divisions of Epilepsy: Grand Mal, Petit Mal, Mental Epilepsy.—Tonic and Clonic Spasms.—Epileptic Vertigo.—Cerebral Congestion.—Stomachic Vertigo.—Psychical Epilepsy.—Furor Epilepticus.—Masked Epilepsy.—Status Epilepticus.—Nocturnal Epilepsy.—Ecchymotic Spots.

GENTLEMEN,—We will to-night consider a very important subject,—the disease known as epilepsy. The word is derived from the Greek words *επι*, upon, and *λαμβάνω*, I seize. Epilepsy is an affection of the nervous system. Its study is very instructive, from the fact that it frequently exists in a masked form; therefore its medico-legal relations are very intricate, and, furthermore, not infrequently physicians are led into errors of diagnosis.

X The leading symptoms consist in paroxysmal loss of consciousness, generally attended by convulsions (though the latter are sometimes absent), and followed by more or less profound coma. These are the general leading symptoms. This affection, however, X has one characteristic, unequivocal and pathognomonic,—a feature which is never absent,—one upon which we may always rely, and without which the diagnosis cannot be effected. It is always, therefore, to be looked for, and we cannot too carefully weigh and appreciate it, for it often enables us to make obscure cases plain and apparent. This symptom is abolition of consciousness. I shall frequently revert to this fact while considering the several forms of epilepsy.

The varieties of this affection we will now consider, and you will observe that the phenomena of different cases may be very dissimilar, but that this one particular, unequivocal, pathognomonic symptom is always to be found. Epilepsy is a disease much dreaded by practitioners, mainly on account of its tenacity and disastrous effects. The ancients called it *morbus sacer*, meaning a

sacred disease, which, on account of its remarkable characteristics, they thought was inflicted as a punishment by the gods. It was also termed *morbus demoniacus*, because it was supposed that an individual suffering from epilepsy was possessed by demons; and the reason for this conjecture you will be able to understand better when we come to study the natural history of the disease.

X There is one thing certain about epilepsy: it is one of the most obstinate, indomitable, difficult, and intractable diseases to master. It is a common affection, but one which you will always meet with peculiar apprehension and with little desire to treat. The experienced physician very well knows that the friends or relatives of the patient would rather hear his death announced than that he is the victim of this inexorable disease. This is mainly due to its incurable nature, horrible features, and peculiar tendency to the development of insanity. It certainly has a progressively deteriorating effect upon the mind. The attacks seldom come suddenly, certain warnings generally occurring, which are understood by patients and lead them to be upon their guard. The premonitory signs consist in certain nervous phenomena, existing very often, though not in all cases. When they do occur, it is not always by any particular or distinct manifestation, there being no special or typical warning, but there is generally complaint of a peculiar vague, indefinite sensation, which seems to originate on the periphery of the body with a certain centripetal tendency; that is, it always travels in the direction of the nervous centres. It often occurs in the extremities,—in the hands and feet,—and consists of a tickling sensation, a species of formication: sometimes it is of a burning character. There is no mode of description which can clearly convey to your mind the picture of *aura epileptica*. Of course, these warnings are not always of the same nature: sometimes they are not physical, but are entirely of a psychical character. Tanner relates the case of a patient of Dr. Gregory, of Edinburgh, who at the beginning of an attack would complain of seeing a little woman in a red cloak pursuing and striking at him. This apparition would throw him into a state of great fear and anxiety, which was soon followed by an epileptic paroxysm. Each seizure was preceded by the same delusion.

Before describing the symptoms, I shall say a few words in regard to the natural division of the disease. Epilepsy is divided

into *grand mal*, *petit mal*, and *mental epilepsy*; or, in other words, the greater evil, the lesser evil, and mental or psychological epilepsy.

The *grand mal* generally corresponds to the convulsive form of the disease; the *petit mal*, or epileptic vertigo, to the vertiginous form. The latter is not so violent in the intensity of its manifestations; but authors are unanimous in the opinion that it produces greater impairment of the intellect than the convulsive form. The *mental* or *psychical* form is a variety characterized by the absence of the vertiginous and convulsive developments. It consists of a transitory attack of epileptic insanity or fury. But the prominent characteristic feature of all these forms is one common symptom pervading them, which is absolutely pathognomonic of the disease,—the complete unconsciousness during a paroxysm.

Let us commence with a description of the symptoms of the *grand mal*. Suppose a man to have been seized with it in this amphitheatre: what would you expect to witness? The patient, in the first place, almost invariably falls; but immediately prior to doing so he utters a loud shriek, and at the very moment of the cry, at the very commencement of the paroxysm, there is intense pallor of the countenance, to be followed by lividity, which is one of the ulterior phenomena of the epileptic paroxysm. Hence, when you study the manifestations in the order of succession, you have the shriek, the fall, the pallor, and from the very beginning of the paroxysm there exists a remarkable, profound, and undoubted unconsciousness. Many variations of the latter occur, according to the different forms of the disease, but in the *grand mal* this abolition of sensation is so profound that pinching your patients, sticking them with pins and needles, rough handling, or exposure to any painful sensation, even a hot iron, will elicit no response, no evidence whatever of its appreciation. So true is this, that a person who falls during an epileptic fit is always in danger, for wherever he may chance to fall there he will remain until the fit terminates. Were he to tumble into a grate full of burning coals he would remain there, entirely unconscious, till the paroxysm ceased. This liability is necessarily a constant source of danger to the patient, who may seriously injure himself, and is therefore a constant source of anxiety to his friends. This is quite different from what occurs

in hysterical women, who generally manage to select some soft spot upon which to tumble. After the patient is on the ground, and during the continuance of the pallor, there is throughout the entire muscular system a condition of tonic spasm. As you will frequently in these lectures hear the terms tonic and clonic spasms, I shall proceed to explain their meaning. By tonic spasm is meant a spastic non-relaxing or tetanic rigidity of the muscles involved; while in clonic spasm there is a certain alternation of contraction and relaxation, of flexion and extension, of the muscle. In tonic spasm there is a retention of rigidity, a continued contraction of the muscular fibres, which remains uninterrupted till the spasm passes away. In studying the phenomena of epilepsy, we must of necessity pay considerable attention to these two forms of spasm,—the tonic and the clonic.

Now, in the commencement of the paroxysm of *grand mal* we have tonic spasms. But these last only a short time; indeed, they could not well be prolonged without causing the death of the patient. There is then always danger of asphyxia, the pectoral and other muscles of respiration being in a state of unyielding contraction; and necessarily there is an undue pressure upon the blood-vessels, preventing the free circulation of the blood, and but for their short duration, usually not more than thirty seconds, and the timely occurrence of relaxation, the patient would inevitably perish. But after the rigid tetanic contraction we notice a relaxation, an alternate flexion and extension, of the voluntary muscles; and this latter feature constitutes a marked character of epileptic convulsions. During this period all the limbs work spasmodically, the eyeballs are fixed and the pupils dilated, the muscles of the face jerk and twist, froth escapes from the mouth, and, in consequence of the disturbed action of the facial muscles, the jaw is spasmodically closed, while the tongue protrudes, causing the biting or laceration of that organ, and the saliva is tinged with escaping blood. Now, the laceration of the tongue is often but not invariably present; and some pathologists, especially Schroeder van der Kolk, attach great importance to this symptom, which, however, would be most valuable should you be in doubt as to whether the case was one of epilepsy or one of hysteria. "Thus," says Van der Kolk, "we must infer that in epileptic patients who are accustomed to bite the tongue in every attack, the irritation and vascu-

lar dilatation are more decided in the tract of the hypoglossus and corpus olivare ; in the epileptics, on the other hand, who never bite the tongue, these changes are better marked in the course of the vagus." Often during the continuation of the convulsions there is a relaxation of the sphincter muscles, followed by an evacuation of fæces and urine. This often furnishes evidence of epilepsy, especially if at night a person wakes and finds his bed soiled or wet. Trousseau considers this a diagnostic point in nocturnal epilepsy, which will reveal it when we are otherwise unaware of its existence. The convulsions now pass off, to the great relief of the spectator, for they are terrible to witness, something to which I never could accustom myself, the distorted features, livid face, bitten tongue, bloody froth, terrible agitation of the whole system, and fearful expression of the countenance, making the whole a condition so appalling as to leave a painful impression upon one's mind, no matter how slightly sympathetic he may be, and justifying the old name of *morbus demoniacus*.

After this state has passed away, the patient becomes comatose. It is not a deep, long coma, but a drowsy stupor, lasting half an hour, sometimes one or even two hours, as the case may be. There is occasionally, although rarely, a complication of paralysis, and the patient may be hemiplegic ; but always recollect that the hemiplegia of epilepsy is invariably transient, lasting, at most, three or four days, though very exceptionally that long ; whereas in cerebral hemorrhage the hemiplegia is generally permanent, lasting several months and usually for life. Now, never overlook the fact, which many physicians ignore, and to which Dr. Todd was the first to draw attention, that transient hemiplegia often complicates an epileptic paroxysm.

The second form is *epileptic vertigo*, which it is necessary for you to understand thoroughly. I wish by all means to impress upon your minds the salient features which bear directly upon its diagnosis, for it is often misunderstood by physicians, and is the field in which specialists are eminently successful in diagnosing, because they do not ignore the natural history of the malady. The expressions epileptic vertigo, vertiginous epilepsy, *petit mal*, are all synonymous. If some patient would, just for our convenience, come before us and fall into *petit mal*, it would be an easy matter to demonstrate some of its appearances. But, unfortunately, this

form occurs when we do not see it, and it is, nevertheless, just as important to recognize its features as those of the more apparent convulsion of *grand mal*. Any old woman knows when a person falls into a fit, but it takes a well-informed physician to recognize a case of epileptic vertigo, for this may occur with very slight manifestations. A person might be on trial for his life, the defense might plead epilepsy, and the accused might have an attack of epileptic vertigo in open court and no one present be aware of its occurrence. From this you see the great importance of being on your guard, for if not vigilant you may fail in the diagnosis of this complication. You might regret, when too late, not having been able to save a person's life; or, taking a more selfish view, the right diagnosis may help you to build up your reputation in medico-legal cases. You must, therefore, make yourselves familiar with all the facts connected with this form of disease, which is one of the worst affections to which mankind is subject. These considerations are not a study for the specialist alone: they deeply interest all practitioners, and as future physicians it behooves you to recognize epileptic vertigo even where it occurs in a slight form. You remember the characteristic symptom, loss of consciousness. This may be more or less profound, more or less apparent. Should a man in an epileptic fit fall into a grate filled with burning coals and remain there convulsed for a short time, the unconsciousness would be very evident. But how is it (as related by Trousseau) that a musician playing the flute in an orchestra may be suddenly seized by a short vertigo, but still the automatical movements of his fingers may be continued just as they were when under the direct control of the will, and the person has all the time been playing in perfect accord, without missing a single note? Yet he has had an attack of epileptic vertigo! What would you think of a man who, while playing cards, just as he holds up one ready to place upon the table, suddenly gives a vacant look, a peculiar stare, a sigh, and down comes the card, and he plays on as usual? This man has also had an attack of epileptic vertigo. Or, to cite another instance from Trousseau, an architect might be walking upon the floor-joists of a building in process of erection; you might notice him suddenly acting strangely; a grimace or twitching of the countenance might be observed; he might shout, and go on without falling,

though for a moment he was entirely unconscious, and only automatically retained his equilibrium; still, the individual would remember nothing that occurred in this short attack, which only lasted a few seconds. This again would be epileptic vertigo. Or a man may be seated at table, raising his fork to his mouth, and suddenly we notice a vacant look, a statue-like appearance, a wild, unnatural stare; the fork drops; there is a pause, and then he may resume his dinner and continue eating as before this short seizure, which was epileptic vertigo.

It is impossible for me to describe the different phases, varieties, shades, and imperceptible diminutions of intensity of one form of epileptic vertigo as compared with others; some being more apparent than others, and many almost imperceptible in character. What I take pains to impress you with is the peculiar grouping or blending of the phenomena; and these few illustrations will enable you more readily to seize upon the distinctions and comprehend the varying aspects of the disease. In some forms there may be a temporary fixation of the eye, meaningless exclamations, a peculiar appearance of the countenance, evinced by a slight convulsion, superficial twitching of the facial muscles, all of which are transient phenomena. Why do I so strenuously insist upon the recognition of epileptic vertigo? Because in the latter, as in many other brain-diseases, cerebral congestion is the convenient explanation resorted to for the want of a more definite term wherewith to conceal professional ignorance and neglect. This assumption generally is plausible from the fact that it is not always easy to controvert. By way of illustration, suppose a man who has occasional attacks of dizziness consults his physician, stating that he loses his consciousness for a few moments, that he has no particular recollection of what occurs during the paroxysm. Congestion of the brain suggests itself immediately to the doctor as the solution of the difficulty, which without further consideration he hastily adopts. A second individual has transitory spells of dizziness, with some trouble of the internal ear, and at times feels faint; the diagnosis in this case is without hesitation stated to be cerebral congestion. A third presents himself feeling faint, weak, and dizzy, with painful sensations in the stomach, eructations, pyrosis, gastralgia, and many other dyspeptic symptoms, affording to the mind of the physician grounds by which he arrives at the

inevitable conclusion of cerebral congestion, which is saddled with all imaginable pathological perplexities.

Now, the purpose I have in view in describing these three different conditions is to show you how to recognize pathological states occurring in every-day life, having, not, as stated above, a common, but, on the contrary, a most varied origin. The first patient had no congestion at all; he spoke of his attacks of unconsciousness, and in all probability had epileptic vertigo. The conclusion is obvious, for in cerebral hyperæmia the symptoms are not so transient. The dizziness and unconsciousness are not momentary. Are attacks of congestion ever so short in duration as scarcely to elicit notice before disappearing? When speaking of hyperæmia of the brain, I repeatedly told you that we have first the symptoms of irritation, and that these are oftentimes followed by those of depression. In fact, there is no hyperæmia which exists but for a few seconds. If you ask some ignorant or careless physician to explain the remarkably short duration of the attack which he termed congestion of the brain, he will gravely assert that that organ is liable to fleeting congestions. This person, therefore, had an epileptic seizure; it was transient in character, and hence not a congestion. Or a man may fall and be unconscious without any apparent convulsive seizure; a physician happens to be present, and an apoplectic attack is immediately diagnosed. The omnipotent lancet is resorted to, and at once the patient rises and walks, affording a living illustration of the efficacy of venesection! The physician says, The man evidently had a clot in the brain, and has been immediately restored! But there was no clot: the patient simply experienced an attack of epileptic vertigo, which caused the fall and the temporary loss of consciousness and of motion. Now, as a rule, when seized by a fit of vertiginous epilepsy patients do not fall; they generally grasp some near object by which to maintain their upright position. But why was this not a case of cerebral congestion or of cerebral hemorrhage? Because the phenomena were altogether too transient, and their evanescent character was just the peculiarity of epileptic disease. It would be an extraordinary circumstance for a clot to knock a man down for only a few minutes; a very strange kind of cerebral congestion of apoplectic character that would fell a man to the earth and a few minutes afterwards allow him to get

up and attend to his business. It was an epileptic seizure; and the physician did not recognize it because he attributes everything to cerebral congestion, and has not studied the true nature of the affection we are now considering. To the venesection was attributed the resuscitation, whilst in fact its results were negative. Now, this brings us back to the second illustration which I mentioned. It had nothing in common with cerebral congestion; neither was it epileptic in character, for disease of the internal ear often produces vertiginous spells. But in the third instance we have an interesting condition, which I am now about to describe. You recollect that during my lectures on the diagnosis of anæmia of the brain I referred to stomachic vertigo, which occurs in consequence of gastric disturbances. It is not very long ago that a gentleman was sent to me from a distance in order that I should confirm a diagnosis of cerebral thrombosis, when all he had was stomachic vertigo. Whenever dyspeptic symptoms are associated with vertigo, blame the stomach, but not the nervous system. Hence a mistake is here utterly unpardonable.

You are all anxious to know the distinguishing features of stomachic and epileptic vertigo, when the phenomenon of vertigo exists as the most prominent symptom. Your determination must be influenced in the first place by the history of the case. This will tell you whether a dyspepsia exists, as evidenced by gastric or intestinal disturbances. Next you determine the extent and duration of the unconsciousness. If it be the epileptic form of vertigo, there will be more or less complete unconsciousness; but if it be a stomachic vertigo, this latter symptom will be absent. The fact of the non-abolition of consciousness, the forgetfulness on the part of the patient of everything that happened during the attack, will enable you to arrive at proper conclusions. If you question the patient he will readily answer, "Of course, my consciousness was not impaired; I knew what was occurring, but there was a certain turning around of objects about me which caused me to reel and almost lose my equilibrium." You remember that in epilepsy the peculiar unequivocal symptom which in all forms, shades, and varieties is ever manifest, is the abolition of consciousness: consequently, the patient is unable to remember anything that occurred during the attack. You are now aware of the possibility of averting errors.

We have come to the description of the next form, which is psychological or mental epilepsy. Possibly you have heard of *mania transitoria*, temporary mania or fury, in which a patient is seized with a paroxysm of blind, instinctive rage or maniacal excitement, lasting sometimes one day, sometimes several days, though generally of short duration. I have never believed it to be an ordinary form of insanity, but consider it a latent, masked form of epilepsy. The individual is entirely unconscious of his actions during the maniacal attack. It is really a substitution of epileptic mania for the epileptic convulsion or vertigo. Hence we must distinguish epileptic mania, or *mania transitoria*, from the ordinary forms of insanity. I have already told you how cunning maniacs often are, what a deliberation exists in their acts, and the extraordinary manner of their behavior. They are, it is true, rash and impulsive in one sense, but sometimes they converse rationally and reason correctly, though from erroneous premises, and when they commit a homicide, or other offense, it is frequently the result of a premeditated plan to overwhelm their victim. They display great ingenuity and craftiness; they recollect and talk about the perpetration of the crime and its very details, and even attempt a justification based upon some delusion. But in *mania transitoria* this fury does not occur while the patient is conscious; there is a total oblivion concerning the acts performed during the transient rage. This is a favorite ground for experts on which to defend criminals. I remember a celebrated case that happened in New York, of a distinguished general, in which the verdict was that at the time of the commission of the crime the accused was insane, but that the moment preceding and the moment succeeding the deed he was rational. Now, this would be an astonishing rapidity of change in the mental state, and the plea was not reasonable, its absurdity being equaled only by its boldness. It is quite similar to those forms of so-called cerebral congestion diagnosed by many practitioners, which we have just been discussing, so temporary and fleeting in their nature as to last only a moment and leave no traces, and excited without any apparent cause. But in *mania transitoria* this extreme transiency does not exist; it may last for several hours or more, though generally it is short; but it is never so absurdly brief as in the case to which I have just referred. *Mania transitoria* is generally characterized by great

fierceness and fury ; and when the patient attempts homicide, his rage is usually not expended until several victims have felt its force. The epileptic explosion is characterized by its brutality, the number of blows, and the cruelty and entire want of motive. An ordinary mania has always a delusion for a motive ; but in *mania transitoria* the acts are without adequate incentive : the patient, suddenly becoming enraged, kills his best friends remorselessly, and during the commission of the deed, and afterwards, is perfectly unconscious of his act. The abolition of consciousness and of memory causes this latter affection to be regarded as epileptic in its nature. As future physicians, it behoves you all to know of the existence of this masked form, called epileptic mania, or *furor epilepticus*, and that when it exists the convulsions or vertigo are masked or replaced by this fury.

Larvated epilepsy, as it is called, is most interesting in a medico-legal point of view. For the present it suffices you to know that not infrequently the epileptic manifestations do not occur in the ordinary forms, such as convulsions or vertigo, but are concealed and masked by some influence peculiarly apt to expend its violence through the psychical faculties, accompanied, however, by the same loss of consciousness that pervades all the forms of epilepsy. The paroxysmal recurrence of epilepsy is important ; and you may ask me, How often do epileptics have fits ? how often do these paroxysms of unconsciousness occur, with or without convulsions ? I answer, it may be perhaps only once during a lifetime, perhaps once a year, once a month, or once a week, or it may be every day, or every night, or several times a day.

There may be what is termed the *status epilepticus*, where there are, occasionally, tendencies to the rapid recurrence of the paroxysms, during which the patient passes from one to another. One may have nocturnal epilepsy, in which the fit never occurs in the daytime at all. This fact it is very important for you to know. Perhaps some of you are nocturnal epileptics, not having any proof to the contrary. Let me relieve your minds. If on awaking in the morning you do not find the bed wet with urine or soiled by fæces, you probably are not epileptics. But if persons void their urine in bed, they are not infrequently epileptics. I have had under my treatment for several years a young lady who has had nocturnal epilepsy. When I inquired of the mother if

she were not afraid to let her go to parties, picnics, and other places of amusement, she replied that she had no fears upon that score, as she was never convulsed except at night, to which fact, in eighteen years, she had not known an exception. Now, mothers are exceedingly sensitive upon this subject: they do not like to make any admissions. They will occasionally consult you about a certain form of nervous disease in their children which may be obscure to you, and perhaps you may not exactly understand. When you ask for information they will try to frustrate your investigations, though they have called upon you to seek your professional assistance. This may seem strange to you, because you do not realize what peculiar beings women are; they act rather inconsistently at times. If a mother comes to you with her little daughter and wishes to know what causes the loss of the child's memory, you ascertain the child's exact condition. Loss of memory is rather characteristic of cerebral softening, but the age of the patient would exclude it in our hypothetical case. You observe that the child has an epileptic look, and ask the mother if she has ever had a fit. But here the mother indignantly answers, "Of course not!" The supposition that her lovely daughter is thus afflicted may occasion offense. Forewarned, your pertinacity, however, will not thus be baffled. Try to ascertain the habits of the little patient, and ask if she ever wets her bed. The answer is, "Oh, yes, she does; and has done so for a long time." Well, wetting the bed and progressive loss of memory are two signs that go far to prove that the patient is epileptic, in nine cases out of ten. If you are in doubt, examine the skin, and you will perhaps find little ecchymotic spots like flea-bites, which do not disappear upon pressure, and which you are more apt to find shortly after an attack than later on, but which are quite pathognomonic of the convulsive form of epilepsy, occurring during the muscular contortions by the yielding of a few capillaries, and attended by an extravasation of blood. These spots confirm the diagnosis. Hence, to the expert and the physician who carefully study the phenomena of nervous diseases, loss of memory, wetting the bed, and ecchymotic spots constitute the unerring signs of nocturnal epilepsy, as well as of some other doubtful forms. There is also a peculiar, indescribable, stolid appearance in the countenance of epileptics; an habitual dila-

tation of the pupils, and commonly an intense hebetude manifested in the features. These phenomena you will become familiar with by repeated contact with such cases and by close observation. An expert can go through the wards of a hospital and recognize six out of ten confirmed cases simply by their peculiar *facies*.

It has been stated that there is an hysterical face; but I never could single out an hysterical woman by her expression or countenance alone,—there are so many who are hysterical; and there are no particular features which I consider pathognomonic. This brings us to the diagnosis of epilepsy, which we will discuss in the next lecture.

LECTURE XX.

EPILEPSY—*concluded.*

Differential Diagnosis.—Infantile Convulsions.—Apoplectic Coma.—Influence on the Mind.—Feigned Epilepsy.—Etiology: Hereditary Influence, Transmutation of Nervous Diseases, Physical and Moral Causes, Malformation of the Head, Forceps in Protracted Labor, Catamenial Troubles.—Hysterical Epilepsy.—Pathology.—Told's Theory.—Niemeyer's Theory.—Anatomical Location.—Vaso-motor Nerves.—Prognosis.—Treatment.—Ophthalmoscopic Examination.

GENTLEMEN,—There are several important points which we have to consider in regard to the diagnosis of epilepsy. The first is the differential diagnosis, in children, between epilepsy and infantile convulsions. It often happens that our little patients are subject to certain eclamptic attacks, and the mother of course is anxious to know whether or not the seizure be epileptic in its nature. The only point which can guide you in your opinion is the existence of a tendency to a recurrence of the attacks. This, if it exists, will cause you to apprehend epilepsy. In ordinary infantile convulsions the paroxysms are generally due to some particular cause, such as an overloaded intestine, worms, or difficult dentition. You should always try to ascertain what has led to the attack, and, by removing the cause, endeavor to prevent future relapses.

The coma of epilepsy is sometimes mistaken for that of cerebral hemorrhage. But this blunder you can always avoid by ordinary caution. In epilepsy the coma is not so deep as in cerebral hemorrhage, nor is the respiration so stertorous; and the peculiar flapping of the cheeks, like loose sails,—which I described as peculiar to the latter, and which I told you was due to a paralysis of the buccinator muscles and of the velum palati,—is absent. Then there is a difference in the pulse, which in cerebral hemorrhage is slow, hard, and labored, while in epilepsy it is quick, frequent, and feeble. These features are generally sufficiently characteristic to enable you to distinguish between the two conditions; but where you are not positive as to the true nature of

the coma, do not venture upon an opinion ; wait, and if it be epileptic it will soon pass off, and the hemiplegia—a rare occurrence, if it occur at all—soon disappears; while in an apoplectic fit the coma is profound and of longer duration, and is almost invariably accompanied by a paralysis which lasts a few months at least, and often a lifetime. The diagnosis of epilepsy from chorea and hysteria will be considered when we speak of these latter diseases.

Repeated seizures of epilepsy have undoubtedly a very prejudicial influence on the mind, and particularly do they impair the memory. The intellect is rarely so clear as it was prior to the disease, and in proportion to the increased frequency and severity of the attacks will there be, *pari passu*, corresponding mental impairment and diminution of mental activity. This is the opinion of most authors ; and it corresponds with my own experience. In J. Russell Reynolds's work it is contended that epilepsy does not necessarily produce this effect. However, he stands, I believe, almost alone and unsupported in this assertion. It is true, there are some cases of epilepsy that are not very well marked, and in these cases the mental impairment may be overlooked, if the attacks be light and the intervals between the paroxysms be prolonged. The damage to the intellect will soon be evident in the more frequent cases, and in the vertiginous forms of epilepsy much more than in *grand mal*.

I intended to allude in my last lecture to the fact that the disease is occasionally feigned, and it may become your duty to determine whether it be simulated or genuine. If you recollect the characteristic, univocal symptom of epilepsy, the phenomenon upon which I laid so much stress, which is the abolition of consciousness, you will have no trouble in effecting a diagnosis. Now, suppose you are in doubt as to the character of a fit where a person has fallen without having bitten his tongue, or without involuntary evacuation of faeces or urine (as may sometimes happen in true epilepsy), you will immediately have recourse to the only test, and that is, the presence or absence of consciousness. This you can easily determine, for instance, by pricking the individual with a pin, or by touching him with a hot iron, or by applying to him some irritant. If he withdraws the limb, it immediately shows that the pain has been perceived, and that he does not labor under

epilepsy. Impostors very frequently assume it with a view of speculating upon the charity of the public, and by keeping a piece of soap in the mouth, purposely lacerating the tongue, and falling often, deceive even more than casual observers. The object to be attained is frequently itself a cause for suspicion, as is clearly indicated in the case related by Tanner of a quasi epileptic who walked about the streets of London, and whenever he fell down was particularly careful to have a card lying prominently before him with the inscription, "Do not bleed me; a glass of brandy-and-water will cure me." Now, I am satisfied that you will not allow yourselves to be led astray by assumed epileptics: so we will proceed to consider the etiology or causation of the disease.

In the first place, hereditary influences play an important rôle in the causation. You will often find that the patient's parents, or some of his relatives or ancestors, have been afflicted with epilepsy; and sometimes its existence is the evidence of the transmutation of nervous diseases spoken of by Trousseau, to which I called your attention while speaking of insanity. We have already seen that nervous affections of a certain form are frequently evidenced in subsequent generations by either hysteria, insanity, epilepsy, or alcoholism; and these different varieties may exist separately in various members of the same family. For instance, one brother may be a maniac, another an epileptic, a sister hysterical, etc. Almost any violent moral excitant may become a cause of epilepsy in a patient who is very susceptible of this disease,—for instance, anger, fright, or jealousy. As physical excitants, venery, drunkenness, masturbation, and abuse of the sexual act, even where normally performed, all may superinduce epilepsy in individuals prone to attacks of diseases of the nervous system. A malformation of the head is classed among the causes of epilepsy; and it has been asserted by some obstetricians that the use of the forceps in cases of unusually protracted labor may result in epilepsy in the child. The fear of this result should not, however, prevent you from applying them without hesitancy when necessary. Catamenial troubles are very often the cause of the more curable forms of epilepsy; and we often find amenorrhœa and dysmenorrhœa, or the development of puberty, etc., to have a certain connection with developments known as hysterical epilepsy. By establishing the catamenial discharge, if tardily produced, or by re-establishing

it when suppressed, we often cure the affection. The same good result is often accomplished when the nervous affection exists in connection with uterine diseases, or with morbid conditions of the genito-urinary organs; not by treating the epilepsy alone, but by directing the medication to the other ailments. Moreover, bromide of potassium, which is so valuable a remedy in nervous diseases, is of double efficacy in such cases, especially if they be complicated with diseases of the sexual organs or intra-pelvic troubles.

The pathology of epilepsy is a subject upon which I might discourse at great length, but for want of time I shall merely consider the principal theories in vogue. Dr. Todd considers epilepsy to be due, to some extent, to the influence of certain electrical conditions of the nerve-centres, which, at times, are more or less surcharged with electric or some analogous force, produced by a toxic condition of the blood and discharging itself in explosions, thereby relieving the undue and accumulated tension of nerve-force with which they are pervaded. Niemeyer calls attention to the fact that epilepsy is one of the very few diseases in which the convulsions and paralysis occur simultaneously. Of course you all understand that by convulsions are meant the clonic spasms as they occur in the *grand mal*, and that by paralysis is meant the psychical condition corresponding to the abolition of consciousness always present in all forms of epilepsy. This loss of consciousness, or psychical paralysis, is produced by a sudden shutting off of a quantity of blood from the brain. There seems to be an irritation of the vaso-motor nerves accompanying the cerebral blood-vessels, in consequence of which the latter immediately contract, diminishing their calibre, and the result is an intense anæmia of the brain. This explains the mental paralysis, a symptom of depression evinced by the phenomenon of loss of consciousness. It is the closure of the arterioles supplying the brain which produces this temporary anæmia, keeping pace with the loss of consciousness; and *pari passu* we have convulsions, first the tonic, then the clonic spasms; and thus in epilepsy we have first the paralysis (mental) co-existing with convulsions of the voluntary muscles. The subsequent clonic convulsions are stated by some authors to be the result of the excessive venosity of the blood, superinduced during the violent spastic muscular contractions in the earlier moments of the attack.

Schroeder van der Kolk, a most shrewd, acute, and remarkable observer of nervous diseases, was one of the first pathologists who taught the anatomical location of epileptic affections. He maintained that it is a disease of the medulla oblongata; that, moreover, it is seated in certain ganglia of that organ; and to these parts he directs you, in your post-mortem examinations, to seek for characteristic tissue-changes. Several authors, however, contend that epilepsy is not an organic disease; that it is merely a neurosis, without any constant anatomical characters, and leaving no post-mortem changes at all. Sometimes this is true, as when the epilepsy is only of short duration. Here, probably, we have no evidence of textural alteration or histological changes in the elements of the medulla oblongata; but where the disease has been of sufficient severity and of a long duration, they are nearly always apparent. Quite a difference of opinion has existed among authors as to whether the true seat of epilepsy is in the medulla oblongata proper or in some ganglionic structures or nervous centres situated upon the medulla, which, though in close relation to it, belong to the great *sympathetic* system of nerves, and, notwithstanding their anatomical location, do not belong to the cerebro-spinal axis at all.

Most modern authors agree that these ganglionic cells, situated in the medulla oblongata, are intimately associated with the vaso-motor centres of the entire human body. It is true that this is denied by Brown-Séquard, who recently has contended that these centres are situated in the pons varolii; but concurrence of testimony is against him. Now we can understand how it is that in epilepsy we have always the pathognomonic, univocal symptom,—the abolition of consciousness. It is because the vaso-motor nerve-centres are irritated, and by morbid innervation propagate this irritation to all the cerebral vaso-motor nerves, causing a contraction of the cerebral blood-vessels, followed by an intense anæmia and characteristic mental paralysis. In consequence of the changes in the medulla oblongata, we find thickenings, indurations, and vascular alterations as the disease advances and becomes more evident, not in recent but in confirmed cases. In old cases, in consequence of the continued irritation, these textural changes are more apparent, for, as direct consequences thereof, we have increased vascularity; the liquor sanguinis exudes from the tortuous vessels pressing upon the nerve-centres, thereby causing

the changes in the nervous constituents of the medulla. There is no necessity of entering further into the anatomical appearances of epilepsy; sometimes we find them, and sometimes we do not; but most authors admit that it is the ganglionic cells situated in the medulla oblongata that are at fault. Echeverria says, in his excellent work, "Were I to present a definition embracing the principal features of the disease, I should say that epilepsy is a disease constituted by chronic paroxysms, excited upon a direct or reflex action of the medulla oblongata, in a condition of exalted irritability, coincident with sudden depression in the cerebral circulation and with loss of consciousness, with or without muscular spasms." This seems to be coincident with the manner of causation, for instance (as I omitted to tell you while speaking of the causes), when epilepsy is produced by reflex action, as from worms in the intestines, spiculæ of bone pressing upon a nerve, neuromata, or injuries of some parts of the nervous system.

The prognosis of epilepsy is unfavorable. It is an extremely unmanageable affection; so much so that, with a vast experience, a large and accumulated observation, I admit that I cannot record the cure of a single case of confirmed epilepsy. This is a statement not encouraging, but it is true. In epilepsy, as in insanity, the disease is curable in proportion to the shortness of its duration. But when the case has been one of long standing, passing from practitioner to practitioner until by the force of morbid habit the textural changes have become confirmed, you cannot expect to cure, but only to palliate. Schroeder van der Kolk maintains that just in proportion as the manifestations during epileptic disease are deferred do they become dangerous in character. Now, whatever be the cause, it is a fact that this disease is cumulative in character,—that if no paroxysm has occurred for a long time the discharge of the pent-up force is sometimes accompanied with serious danger, and that the severity of the attack is related to the length of the intervals between the paroxysmal explosions.

The treatment but too often is necessarily merely palliative: we must endeavor to prolong the intervals so as to break up the force of the morbid habit of the recurrence. Now, as regards medication, you can make up your minds that the greater the increase of therapeutic measures for a disease, the more hopeless will it

be. Or, to state this in a different manner, the less a disease is amenable to treatment, the greater is the number of valuable remedies. In epilepsy there are so many medicines recommended that it would be impossible to enumerate them. I shall simply describe those upon which you may most rely. First of all, and the remedy *par excellence*, is bromide of potassium. This medicine most frequently cures recent cases, and is the best palliative in incurable epileptic affections. Next comes bromide of sodium, also a valuable agent, and one which I often prefer upon the ground recommended by Dr. Clymer, that the sodium salts are not foreign to the human organization, like those of potassium. This is the reason why bromide of potassium is more depressive and less liable to be borne well for a length of time by the system than the bromide of sodium. When you give these remedies, give them boldly, and in large and continuous doses, as long as the patient can possibly bear them. I have some patients under treatment who have been taking bromide of potassium for four or five years without any evil results, and I intend to give it to them as long as I continue to treat them, unless contra-indications should arise. Again, bromide of potassium, in proportion as it is freely administered, may produce what is called the bromic cachexia, consisting of certain gastric derangements, a peculiar odor of the breath, dryness of the tongue, the eruption of boils, etc. Some physicians aim at producing this cachexia, asserting that by this mode of treatment they succeed in palliating the disease to a remarkable extent. But let me caution you not to be rash in this respect, as I know of fatal consequences following the reckless administration of the bromides after the development of well-marked cachectic symptoms. Dr. Hammond finds bromide of potassium to be more applicable to, and efficient in, the treatment of the diurnal than of the nocturnal forms of epilepsy. The nocturnal and non-convulsing forms, he thinks, are better controlled by strychnia, giving it judiciously to such an extent as to produce gradually its physiological effects. I might speak at length of the bromide of potassium, but shall only say a few words in regard to its administration. For adults, never commence with less than twenty grains three times a day; and should you ask me how much I give, I would tell you from twenty to forty or even sixty grains *ter in die, pro re nata*. Nitrate of silver is a remedy

which formerly enjoyed a high reputation. I have no comments to make upon it, except that I regard it as a positive failure, and, besides, its continuous administration gives an indelible blue discoloration to the skin. The salts of zinc, especially the oxide, valerianate, and sulphate, seem to have beneficial effects. The French recommend belladonna and atropia as efficient remedies; but these are especially dangerous, and liable to produce accidents unless a person be an expert in their administration and have his patient under continuous observation. When you give a preparation of belladonna, I would advise you never to give atropia, but only the solid extract of the drug. Ergot has of late obtained quite a reputation in the treatment of epilepsy. It probably acts upon the ganglionic centres of the sympathetic system. Some excellent articles have appeared of late in the "American Journal of Insanity," wherein it is claimed that ergot has proven wonderfully successful in the treatment of some very intractable forms of epilepsy, where other drugs had completely failed. I believe that this medicine may be very favorably mentioned, and deserves to be tried, especially in combination with bromide of potassium. Recently I had a most satisfactory result from its use combined with bromide of potassium, in the case of a young lady treated ineffectually by distinguished physicians in this country and in Europe for epilepsy, having had several attacks monthly for the past twelve years. Since she has been under the ergot, which I tried while almost in despair of even being able to palliate the affection, she has had but one or two paroxysms in the past nine months. When the first symptoms of bromic cachexia occur, immediately diminish the dose, for that is the time particularly appropriate for the administration of tonics, especially the mineral tonics, such as the chalybeate preparations, which you may combine with cod-liver oil. Dr. Chapman, of London, claims to have produced remarkable cures by means of spinal ice-bags. This plan I have tried, perhaps not so thoroughly as necessary, but in my hands it has failed, and I cannot attach to it the importance given to it by some authors. Baker Brown, who has paid great attention to diseases of the genito-urinary organs, finds a fruitful cause of epilepsy in nymphomania, or irritation of the sexual organs attending erotism, and contends to have ascertained in such cases a most remarkable means of cure in the excision of the clitoris.

In other hands, however, the results have been so futile and unavailing as to justify the consigning of this operation to a well-deserved oblivion. Schroeder van der Kolk holds that the use of counter-irritants, such as issues, dry cups, or setons to the nape of the neck, often exerts a beneficial influence in the cure of epilepsy; and this would seem reasonable, on account of the vicinity of that portion of the neck to the medulla oblongata. But in these days of refinement patients rebel against the use of issues and setons, which are very liable to become offensive, especially in warm weather: dry cups, however, are to be recommended.

Besides the use of medicines, always keep the head in an elevated position and the bowels regular: make the patient sleep on a hard mattress, and pay attention to his hygienic conditions. In epilepsy, as in all other nervous diseases, place your patient in the most favorable hygienic condition, as there is always a tendency to depression and prostration of the vital powers. Therefore administer tonics, which form an important element in the treatment. The nitrite of amyl is a remedy attracting at present much attention. Dr. J. T. Dickson, in his work on "Medicine in Relation to Mind," states that "nitrite of amyl is a drug I have used in epilepsy with eminent success, and I believe that further observations will prove it to be a most valuable medicine." Dr. S. Weir Mitchell, of Philadelphia, in a recent paper read before the Philadelphia College of Physicians, extols this remedy. The importance of his observations is such that I shall quote them at some length. He says, "For more than a year I had been aware that nitrite of amyl would be a proper means to use in epilepsy. It was clear to me that nitrite of amyl caused with rapidity fullness of the vessels of the whole head, and that near to the onset of an attack of epilepsy there is a condition of vasal spasm. I hoped that I would be able by the use of the nitrite to counteract this state of vascular contraction, and so to break the chain of morbid phenomena, and thus end the attack before its more disastrous consequences should follow. This reasonable expectation was not disappointed. I was, of course, well aware that in most cases of epilepsy there would be no time to secure the inhalation of enough of the nitrite of amyl to produce an effect, but I was also aware that in at least two classes of epileptics the opportunity for its use, would be given. There are rare examples of epilepsy in which

the warning of the coming on of an attack so far precedes the spasm and loss of consciousness as to enable the patient to inhale the nitrite. In other cases the patient has a succession of fits within a limited space of time, and, being then of necessity in bed, is so placed that a watchful nurse may find time to use the nitrite. I waited long for my first chance, but in March, 1872, the opportunity came." (Philadelphia Medical Times, April, 1872.)

"J. C., aged twenty; epilepsy due to venereal excesses; the fits being always preceded, except on two occasions, by spasms of the left hand and arm. As a last resort, three or four drops were put into a vial, and he was directed to inhale it by putting the open vial up one nostril, while with one finger he closed the other, and then made a few full inspirations. The first attempt failed, because, as he said, the spasm of the left limb made him nervous. On the second occasion he began to breathe it the instant the fingers twitched, having pulled the cork of the vial with his teeth. In a few moments he felt his face flush, the carotids beat violently, his head felt full, and the spasm ceasing, the attack at once, and for the first time in his experience, was cut short. Four days later he thus cut short another attack; and the experiment has since succeeded in eleven fits, and failed, from too late use of the nitrite, in two. Moreover, the attacks have lessened in frequency, and now come on only once in ten or twenty days. Not only is there no evil effect from the drug, but his memory has improved; is again taking bromide of lithium.

"During the last two and a half years he has had only seven fits, the last being nine months ago. I said seven fits, but, in reality, only one fit, all of the others having been cut short by the nitrite.

"Since this case demonstrated for me the remarkable power of this agent to check spasm, I have given it for that purpose a number of times, its value being limited by the rarity of the cases in which there is time to secure its full inhalation. In some of my examples the chance for using it has been occasional only, not all the attacks affording the time needed to secure its value. . . .

"In the following case there was a gastric aura, which preceded the fit by an interval so long as to enable the sufferer to inhale the nitrite:

"Miss E., aged twenty-six; has had epilepsy seven years. Her

whole history it is needless to relate. About one minute before the fit comes on, Miss E. has a sense of what she calls 'goneness' at the epigastrium. This sensation passes into nausea, and apparently the fit interferes with the consequent vomiting, which very rarely follows.

"This form of aura is certainly rare. The nitrite of amyl instantly arrests both the nausea and the subsequent fit; but the sense of fullness in the head so alarms Miss E., who is a highly nervous and emotional person, that she is very averse to using it."

In case of possible specific contamination, an energetic anti-syphilitic treatment is to be instituted. When the bromides are used, the addition of bicarbonate of potassium to the solution containing them is advisable, as a means of preventing the irritating effects otherwise produced on the gastric mucous membrane by the liberation of free bromine. These latter considerations probably explain the addition of the iodide of potassium and the bicarbonate of potassium to the celebrated formula of Dr. Brown-Séquard. In conclusion, gentlemen, no matter how obstinate the case you have to treat, be energetic and persevering, and you will often palliate, though you are powerless to cure, one of the most distressing affections with which man can be afflicted. Cases of anæmic epilepsy, in which bromide of potassium would necessarily be not only unavailing but injurious, can best be diagnosticated by ophthalmoscopic examination, which latter procedure is very efficient in aiding our investigations in quite a large number of affections of the nervous system.

LECTURE XXI.

EPILEPSY IN ITS MEDICO-LEGAL RELATIONS.

GENTLEMEN,—It is only of late years that epilepsy has received at the hands of writers upon forensic medicine the attention which its importance demands. The criminal records of all civilized countries may be examined, and the cases wherein this common affection has been considered in fixing the responsibility of the accused will be found few in number, and in too many instances small appreciation has been shown of the grave questions involved in the study of epilepsy,—a disease protean in form and easily misunderstood.

The older cases of Tyler, Bethel, and Winnemore, discussed in Dr. Ray's great work upon "The Medical Jurisprudence of Insanity," bear witness to the fact of legal ignorance respecting this disease, even at a comparatively recent period; while the later cases of Max Klingler and David Montgomery show how great has been the advance of a portion, at least, of the medical profession in knowledge of the affection, and, at the same time, the slow pace at which the legal follows the medical profession where questions of criminal responsibility are involved.

The case of Max Klingler having come under my immediate observation, and embracing all the prominent points which are likely to be present in epileptic cases subjected to legal investigation, I shall offer it to you as a text, and follow it with remarks by way of commentary.

The general facts of the case are as follows. Max Klingler, a boy about eighteen years of age, was apprenticed to his uncle, Henry Weider, a tailor. Upon the morning of the day preceding the homicide his uncle had reproved him concerning his work, and also in reference to the removal of a pistol from a drawer. In the morning, while Weider was making a fire in the stove, Klingler approached him from behind, and, putting the muzzle of

the weapon close to his head, fired, with fatal effect. Mr. Weider's wife, upon rushing to the scene, was seized by the infuriated boy, and struck several times upon the head with a hatchet. Leaving her in an insensible condition, he fled, taking what money there was in the drawer. He ran to the Pacific Railroad depot, and was in such haste that he did not stop to pick up his hat, which was blown from his head in the street. Taking the westward-bound train, he was arrested, several hours after its departure, at St. Aubert station, by Marshal Laibold, who testified that at the time of his apprehension his hands were still covered with blood, and that even his clothes were sprinkled with the damning evidence of his guilt. At the coroner's inquest Klingler confessed the homicide. When he was confronted with the body of the deceased at the inquest, he wept bitterly. Shortly after his arrest he made a statement, in which he admitted that "he had killed his uncle because he made him angry, and was not pleased with his work." *He said he "had made up his mind on the previous Saturday to shoot him."* He also added that "when he came down-stairs the morning of the murder, he bade his uncle good-morning, as usual, but received no answer." He then opened the shutters. When he came back, he saw his uncle making a fire, and, without saying anything further to him, he shot him from behind. He further stated that, "when coming down-stairs, he had no intention of shooting him, but had loaded the pistol in the garret."

The foregoing is a synopsis of the facts as developed at the inquest and during Klingler's first trial.

The case was duly tried, and the defendant convicted. A new trial was granted because of some informalities in the first judicial proceedings, and also on account of the reception by his counsel of depositions from Germany, concerning a fall which the prisoner had received in early childhood. While swinging in a barn he fell from a height of thirty feet, producing a depression of the skull, which is still quite apparent. He was found insensible, "weltering in his blood."

Without further referring to these depositions, it may be stated that they proved:

1. The existence of habitual paroxysms of epilepsy in Klingler's mother and sister, and insanity in a daughter of a maternal aunt.

2. That Klingler himself had been subject to "fits" ever since his fall.

3. A shoemaker in Germany—a disinterested party—testified, also, "that Max was treated by medical men, and I observed, on later occasions, that he suffered from temporary insanity."

In a letter from his sister are found the following pertinent sentences:

"Oh, God! if we could have had but the slightest belief that you could fall into such misfortune on account of your sickness, we would never have suffered you to go to America."

Having established the fact that Klingler was an epileptic, we will now quote a few words from a curious autobiography, written since his incarceration:

"This case would not have happened if I had not received the *sickness* just on the 25th of November, for I didn't think I would get the sickness on account of all these troubles. I had often said it in German that I would get the sickness about this time, and nobody should enter the room until I should unlock the door myself, for I get so crazy that I do not know what I am doing. It is dangerous for any one to be seen by me when I get the sickness. The doctor (in Germany) even said to my parents at my examination that I would become dangerous during my sickness. . . . Oh, I am very sorry that I was so unfortunate, dear friends. I had the sickness on the 25th of November, and, unhappily, the boss entered. I saw him; he came towards me and looked at me, when I struck at something. Then I saw that he wanted to come at me, and wanted to hold me: so I got so intensely crazy that I did not know what I was doing. How I got the revolver in my hand I do not know, nor how he lay there. Then she ran towards me, when she found that he lay there, and wanted to strike me. I did not perceive with what I struck her, and when I came to my senses I saw the misfortune and was scared. Then I left immediately. If I had intended this I would have sent my clothes to some place. . . . I kept the pistol with me every morning, because we had in our neighborhood about ten Indians. Every morning I was first in the store, and was afraid of these fellows."

(This latter circumstance was corroborated by the evidence of Mrs. Weider.)

At the close of the second trial the jury failed to agree, standing seven for conviction and five for acquittal. He was tried for the third time, and convicted of murder in the first degree, and was sentenced to suffer the extreme penalty of the law. After several reprieves, his sentence was commuted to ten years' imprisonment in the penitentiary.

That executive clemency was not misplaced will be acknowledged, we think, when all the features of the case are carefully examined and compared with others of the same character. The main points, as I now present them, were submitted to the Governor of Missouri, and may have had some effect in determining his final decision.

That Klingler was an epileptic, that his disease was the result of a traumatic cause, and that there was a *hereditary predisposition* to this affection,—his mother, sister, and some other relatives having been thus affected,—were all facts proven by the depositions taken in Germany and admitted by the court in evidence. Moreover, insanity had previously existed in the family, a daughter of one of Klingler's maternal aunts having been insane. There remains for me only to state some general facts connected with the literature of epilepsy before summing up the evidence which entitled Klingler to an acquittal and attempting to solve some of the difficult problems presented by this interesting case.

Every tyro in medicine is acquainted with the fatal influence of epilepsy upon the intellectual faculties. How fearful is this scourge, which topples over the lofty edifice of the mental powers, bearing, as they do, the peculiar stamp of that Divine Being "after whose likeness man was created"! These unfortunates are subject, during the *intervals* between convulsive attacks, to a peculiar mental condition, most significant in its tendencies and consequences. Sometimes they are querulous, and seem impelled to commit acts of violence, or have explosive manifestations of most terrific and sanguinary fury.

Trousseau quotes Falret, thus: "This irregularity in the state of their feelings and the degree of their intelligence is necessarily reflected in their talk and in their acts. Hence the excessive variability of their behavior towards those about them. For a certain period of their lives they are laborious, punctual, attentive to the duties of their profession, obedient, and docile, and those who live

with them, or who employ them, find their intercourse agreeable, or are pleased with their services. But at other times their conduct becomes *suddenly* modified, and presents the greatest irregularities. They are then incapable of fulfilling their duties, become negligent, lazy, and indolent. They forget the most elementary things, waste their time, or wander here and there, without aim or object in view, and are themselves conscious of the vagueness and confusion of their ideas. The most deplorable tendencies and the worst inclinations develop themselves in them at the same time: they become liars and thieves; they pick quarrels with those around them, complain of everything and of everybody, are very easily irritated for the slightest cause, and frequently commit sudden acts of violence, which, in most cases, have not the excuse of provocation on the part of the victims of these acts."

The highest medical authority vouches for the existence of epileptic delirium allied somewhat to the phenomena of somnambulism, in which the patient is not totally unconscious, as he is during the more common epileptic seizure, but has a vague, indefinite, dreamy realization of his condition or of passing events. His perceptive faculties may remain active under these conditions, yet his highest intellectual powers, and especially the volitional, are not exerted: hence his actions are, to a great extent, instinctive, or, in some instances, purely automatic. These attacks hold "an intermediate place between simple epileptic vertigo and the convulsive fits." It is not difficult to appreciate the existence of instinctive actions, or even of actions springing from a still lower intellectual source, or, indeed, resulting from purely material causes. There is reason to believe that sensations can be transmitted to and received in the *tuber annulare*, and movements result therefrom, and yet no true idea be developed. Ideas, which are purely mental operations, must originate in the cerebral hemispheres; for it is an undisputed physiological fact that "mere sensation and volition may exist independently of any intellectual action, as they may also exist after the cerebrum has been destroyed."

Volition without intellectual elaboration—a faculty by which an act is deliberately accomplished without the appreciation of the *reason* why and wherefore, without the co-operation of an active intelligence—has its seat, probably, in the ganglion of the *tuber*

annulare. Actions which are perfectly free and responsible are conceived, scrutinized, analyzed, and determined upon in the ganglionic cells of the gray matter of the cerebral hemispheres before the will-force, which is deliberative, determinative, and free, issues its mandates. These mandates are conducted along the tubular fibres—which are the telegraphic media of the mind's wishes and intentions—to the subservient voluntary muscles.

Before proceeding further it may be well to understand the meaning of the term responsibility, which occurs so often in this and preceding lectures. Dr. Bucknill's conclusions express our own ideas upon the subject, and are as follows:

“Responsibility depends upon power, not upon knowledge, still less upon feeling. A man is responsible to do that which he can do, not that which he feels or knows it right to do. If a man is reduced under thralldom to passion, by disease of the brain, he loses moral freedom and responsibility, although his knowledge of right and wrong may remain intact.” *

As an evidence of the most important fact that “the disturbance of the reason which follows a convulsive fit, and especially an attack of vertigo, is not always recognized so easily as it might be supposed,” Trousseau cites the fact that “a medical man, for instance, is sent for to see an epileptic immediately after an attack. The patient answers questions pretty well to the point, follows out the doctor's prescriptions pretty accurately, but a few hours later has not only forgotten what occurred during the attack, as the rule is, but he has forgotten all the foregoing circumstances, in which he had apparently concurred with so much presence of mind. It must, therefore, be concluded that his intellect had been deeply perturbed.” Again, we find the same author maintaining that “not only may the patient's reason remain in a perturbed condition for some time after the attack, although a superficial observer may not perceive it, but it sometimes happens that during the attack the epileptic seems to retain enough reason to appear free.”

It is therefore apparent that there is extreme difficulty in determining the criminal responsibility of epileptics, that their mental

* *Unsoundness of Mind in relation to Criminal Acts*, second edition, London, 1857, p. 59.

condition is entitled to the most careful study, and should be regarded with the most elaborate circumspection by medical jurists.

Trousseau establishes the fact by numerous cases that "sudden and irresistible impulses are of usual occurrence after an attack of *petit mal*, and pretty frequently after a regular convulsive fit." He gives the formal opinion "that the patients should not be held responsible for their acts, whether these be followed or not by grave and painful consequences, the gravity of the act itself having nothing to do with the question. The individual is not a free agent for the time, and is therefore free from guilt." Facts cited by such eminent men in science as Baillarger, Boileau de Castelnau, Delasiauve, Echeverria, Gray, Esquirol, Falret, Lagrand du Saulle, Ray, and Schroeder van der Kolk go to support the same opinion.

The sudden outbursts of epileptic fury are so fearful, and sometimes so disastrous in their effects, that no mania is capable of greater and more uncontrollable violence than the epileptic. He is dreaded by all around him, and he may even become his own enemy. His violence is "blind and instinctive," although it appears from careful observation to be responsive to some terrible hallucination. The hallucination is generally forgotten by the individual after the seizure has passed away, but the manner, attitude, gestures, and acts are evidently referable to the fear or horror engendered by some imaginary danger,—imaginary, yet to him a fearful reality.

The most awful and motiveless crimes are perpetrated by epileptics during the existence of this delirium. Its duration is variable, from a few hours to twelve or fifteen days, or even longer. It must be borne in mind that these fits of temporary insanity may be entirely independent of ordinary epileptic seizures,—replacing, preceding, or following them.

Victims to the epileptic influence are apt to be irritated by everything around them, inclined to wander in the streets, and manifest a tendency to obey some concealed, mysterious influence (hallucination) which blindly, yet irresistibly, drives them in a most unaccountable manner, in spite of themselves, to acts of violence. They feel intensely unhappy, consider themselves to be persecuted, and, as is characteristic of other forms of insanity, conceive a remarkable aversion to their friends and relatives, by

whom particularly they believe themselves persecuted and maltreated. "If they have previously harbored any feelings of hatred or thoughts of revenge against any one, these feelings are quickened by their complaint, and suddenly roused to a pitch of intensity which prompts them to immediate action."

Epileptic delusions are oftentimes of a strong religious character; and these at first sight apparently harmless ideas may be the source from which the most sanguinary actions may spring. They are usually of a homicidal character, although self-mutilation or suicide may be the form of culmination.

It is well known that epilepsy soon brings into bold relief the animal traits of character, whose development seems to keep pace with the slow, but generally certain, impairment of the intellectual faculties. Epileptics are commonly inveterate onanists, and the crime of rape or sodomy may often be traced to the salacious tendencies developed under the brutalizing effects of the disease. Of course, where epilepsy of the masked form is pleaded in extenuation of crime of this sort, you should carefully investigate the history of the accused for some evidence of epilepsy in some other form; examine him carefully for vices of conformation, which quite commonly accompany this formidable affection; test his memory, not only in a general way, but also particularly in reference to the act or acts which have brought him under the eye of the law.

An essential characteristic of criminal acts perpetrated by epileptics while under the influence of the special morbid psychical condition is their instantaneousness, their abruptness and suddenness. These features were present in Klingler's case, as we shall presently show. In two cases reported by Dr. Auzouy, where larvated (mental or cerebral) epilepsy was pleaded in extenuation of the crime of sodomy, that learned physician was enabled to determine the fact that epilepsy was not present by ascertaining the absence of these evidences of the disease, together with a perfect recollection on the part of the prisoners of all the incidents connected with the perpetration of the crime.*

Trousseau says, in this relation, "The circumstance that repeated

* *Annales Médico-Psychologiques*, tome xii., Nov. 1874: *L'Épilepsie larvée devant la Jurisdiction criminelle*.

blows are struck, and several wounds inflicted, or several persons injured, deserves to be especially noticed, and seems to characterize the condition of *furor epilepticus*." Speaking of the criminality of epileptics, he uses this strong language: "It may be said, almost without fear of making a mistake, that if a man suddenly commits murder, without any previous intellectual disturbance,—without having, up to that time, shown any symptoms of insanity, and if not under the influence of passion, or of alcohol, or of any other poisonous substance which acts with energy on the nervous system,—it may be said, I repeat, that the man is afflicted with epilepsy, and that he has had a fit, or, more usually, an attack of epileptic vertigo." Again, he says, "Who can calculate the degree of liberty possessed by a man in this state of transition between the actual attack and the complete recovery of his mental faculties? Is there a medical man bold enough to pronounce upon this point, and to affirm that a crime committed after the attack must entail responsibility?"

I would call your attention particularly to the fact that when epilepsy has disappeared for a long time, and is apparently cured, it often breaks forth anew in all its pristine intensity; this statement holds good in regard to each form of the disease. The fallacy of the argument of the prosecution—that because Klingler was not seen in, nor known to have had, a fit for many years, he had not been afflicted for a long time with epilepsy—is patent to all. The possibility, or rather the probability, of his having had nocturnal epilepsy, was not recognized, although a fellow-prisoner who had slept with him testified to the fact that Klingler had acted most strangely, had awakened one night and seized him (the other prisoner) by the throat, and had appeared wild and bewildered. Nor was any emphasis laid upon the fact to the jury, by the defense, that it was quite possible for Klingler to be seized with an unequivocal attack of epileptic vertigo in open court, without an appreciation or recognition of its presence and effect by the judge, prosecuting attorney, jury, lawyers, physicians, or spectators. Again, the fact was entirely ignored that the epileptic paroxysm is not regulated by any legal rule, but that it may occur only once in a man's lifetime, or once a year, once a month, once a week, once a day, or several times in a month, week, or day.

According to Esquirol, in some cases reason returns immediately after an attack, whilst in others it does not return for many hours or many days. In classifying the varieties of epileptic manifestations in three hundred and thirty-nine patients under his care, he speaks of some who had "un délire fugace;" and again, "soixante n'ont aucune aberration de l'intelligence, mais elles sont d'une très-grande susceptibilité, irascibles, entêtées, difficiles à vivre, capricieuses, bizarres; toutes ont quelque chose de singulier dans le caractère." It has been ascertained that when a threatening attack of epilepsy has been averted, its not occurring has given rise, in some instances, to such painful and insupportable agitation as to lead these unfortunates to indulge excessively in alcoholic liquors, or to seek some source of quarrel under which to give vent to their pent-up nervous irritability, and thereby diminish nervous tension; just as does an impatient, hysterical woman when she screams or stamps her foot, with indescribable relief to her morbidly accumulated nervousness.

Ray, in speaking of epilepsy and its legal consequences, in his classical work upon the "Medical Jurisprudence of Insanity," observes, "Another direct though temporary effect of the epileptic fit is to leave the mind in a morbidly irritable condition, in which the slightest provocation will derange it entirely. Sometimes this irritability is accompanied by a sense of anxiety, distrust, jealousy, and unfounded fear, and sometimes by great activity of the lower propensities. . . . Epilepsy seldom continues for any length of time without destroying the natural soundness of the intellect, rendering the patient listless, fretful, indisposed, and unable to think for himself, yielding, without any will of his own, to every outward influence, and finally sinking into hopeless fatuity, or becoming incurably maniacal."

Again, we find the following pertinent remarks of the same author, whose authority in psychological medicine is universally acknowledged, and of whom, as Americans, we may justly feel proud: "To determine exactly the mental condition of an epileptic at the moment of his committing a criminal act is oftentimes a difficult task. It may have taken place in the absence of any observer, in a fit of fury that rapidly passed away, and which, perhaps, may not have followed any previous paroxysm; or the accused, though subject to the disease, may not have recently

suffered an attack, and may have appeared perfectly rational to those around him. . . . Cases of this kind should be closely scrutinized, and where the accused has been undeniably subject to epilepsy, he should have the benefit of every reasonable doubt that may arise respecting his sanity. Less than this, common humanity could not ask; more even has sometimes been granted, under the operation of milder codes than the English common law."

Falls and blows upon the head are well-recognized causes of epilepsy: in Klingler's case there was a well-marked depression of the bones of the skull, evident on inspection. The following from Forbes Winslow's great work upon the "Obscure Diseases of the Brain and Mind" is all that it will be necessary for me to say upon this point: "Repeatedly have I had cases of epilepsy bidding defiance to all treatment—tumors, abscesses, cancer, softening of the brain, as well as insanity in its more formidable types—under my care, whose origin could unquestionably be traced back, for periods varying from eight to ten, fifteen, and even twenty years, to damage done to the delicate structures of the brain by injuries inflicted upon the head."

The grave difficulty, the really serious problem, in Klingler's case, was his avowed *premeditation* to commit the homicide. He admitted to the coroner that he "had made up his mind the night before to kill his uncle." To explain this apparent premeditation there are three possible hypotheses, which I shall consider at some length:

1. This premeditation may have been the result of hallucinations premonitory of an approaching, or the result of a recent, paroxysm.

2. His irritability and peevishness, caused by "an epileptic phase of mind," may have led to premeditation of the deed.

3. The homicide might have been the result of masked, larvated, cerebral or mental epilepsy (these terms being synonymous).

(I.) There can be no doubt that epileptics are subject to hallucinations. It is easy to understand that, owing to some hallucination, Klingler felt a morbid impulse the previous evening to kill his uncle: through the supremacy of his will, coupled with intellectual power not yet wholly subjected, he was able, for the time,

to resist or correct this impulsive tendency. He did not kill his uncle until the following morning, after he had given the usual salutation, and had opened the store for the day's business. Would not an ordinary criminal, premeditating his crime, have perpetrated the act during the darkness and stillness of the night, when his victim would have been completely at his mercy? Would he have courted publicity, by opening the shutters in broad daylight, thereby almost inviting the attention and gaze of the passers-by on a greatly-frequented thoroughfare? What motive existed to incite the boy to such a terrible crime? Not a mercenary one, for he could have appropriated to his own use the small amount of money (thirty dollars) with the greatest facility and with perfect safety without committing the homicide. Again, his uncle—his benefactor, friend, and relative—had always treated him kindly, and only a blind, instinctive, and spontaneous impulse could have urged him on to the accomplishment of this motiveless act of horror. There is ample authority for the statement that total abolition of consciousness in these cases, although usual, is not at all necessary, and that a state bearing some resemblance to somnambulism may exist. Dr. M. G. Echeverria has very clearly elucidated this subject in an article of the very highest value in the "American Journal of Insanity" for January, 1873. He shows conclusively by ample citations from the works of Delasiauve, Legrand du Saulle, Boileau de Castelnau, Trousseau, and from cases met with in his own practice, which has been remarkably rich in cases of epilepsy, that premeditation and action upon such motives as revenge, jealousy, etc., are not at all uncommon in epilepsy.

(II.) As regards the second proposition, I shall not enter into details, as enough has already been advanced upon the literature of the subject to satisfy the minds of most of you. Recognizing, therefore, a frame of mind to which epileptics are obnoxious, which is strange, indescribable, and *sui generis*, the result of a morbid condition over which they have no control, and for which, therefore, they are not responsible, how shall we, or any one, dare to limit the delicate confines where responsibility ends or criminality begins in epileptics?

(III.) Was the homicide the result of mental or masked epilepsy? That an attack of *furor epilepticus* or the so-called *mania transi-*

toria may replace the convulsion of *grand mal* or the vertigo of *petit mal* is now as unquestionable as any other fact in medical science. The labors of Morel, and of his pupil, Dr. P. Geblois, have resulted in a vast collection of facts bearing upon this branch of the subject, the most important branch from a medico-legal point of view. The following case, extracted from an article read before the American Association of Superintendents by Dr. Echeverria, presents all the most important features of larvated epilepsy, and is interesting from its resemblance in many points, especially that of avowed premeditation, to the case of Max Klingler:

"Lasègue, during the recent discussion on the transformations of epilepsy, in the Medico-Psychological Society of Paris, narrates the following:*

"A blacksmith was being helped by one of his assistants in shoeing a horse. Suddenly, without provocation, he struck several blows at the head of his companion, who was holding the horse's foot, and fell furiously upon him. He was arrested, and in answer to the question why he had struck the workman, replied because he could not bear him, and wanted to get rid of him. He pretended that the renewed quarrels they had every day caused him endless difficulties. Upon inquiry, it was ascertained that there was not one word of truth in such a statement. I was directed to examine him, and, being already struck with the rage with which the blows had been given and with the fury he displayed, similar to that peculiar to epileptics, I undertook my researches, surmising the idea of epilepsy. I found nothing characteristic, and did not listen to the system of defense adopted by the prisoner. I had evidently to contend with an individual who had acted under the influence of an irresistible impulse, which he could not account for, and who, on discussing the subject, invented a system to explain it. This man, confined in Mazas, did not exhibit the least disturbance during fifteen days. Suddenly he was seized with the most violent delirium, his strength increased, he tore up the floor of his cell with his hands, and, having detached the hard cement which unites the bricks forming the arched roof of every story, he made an opening large enough to

* *Annales Médico-Psychologiques*, 5me série, tome ix., Janvier, 1873, p. 153.

let himself through, and fell into the cell underneath the one he occupied. He threw himself on the prisoner confined therein, and struck him; a struggle took place, and the keepers had the greatest difficulty to restrain him. He continued for seven days thereafter in a state of constant delirium, the violence of which I could only compare to that of delirium tremens. This unquestionably was an epileptic attack."

In relation to masked epilepsy, Maudsley, in his "Physiology and Pathology of the Mind," makes these observations:

"In such cases there are often sudden and vivid temporary hallucinations. Again, the mental disorder which sometimes takes the place of an epileptic attack, representing, in fact, a masked epilepsy, may appear as simple impulsive insanity. . . . It happens sometimes that the patient succeeds in controlling the morbid idea for a time, calls up other ideas to counteract it, warns his probable victim to get out of his way, or begs earnestly to be himself put under some restraint; but at last, perhaps from a further deterioration of nervous element through bodily disturbance, the morbid idea acquires a fatal predominance; the tension of it becomes excessive; it is no longer an *idea*, the relations of which the mind can contemplate, but a violent *impulse*, into which the mind is absorbed, and which irresistibly utters itself into action." May not Klingler, in his premeditation to kill during an hallucination the night before, have succeeded for a time in controlling the morbid idea, but at last have given way to its excessive tension?

Klingler's intellectual status plainly shows the sad results of the disease. He is sullen, morose, and stolid, and has probably suffered long from *petit mal*, which, according to the high authority of Van der Kolk, "depresses the mental powers much more rapidly than spasms."

Dr. Kelp, of the Oldenburg Asylum, Wehnen ("Der Irrenfreund," No. 1, 1872), cites a case bearing a great resemblance in many particulars to the one under consideration. A young man, R., aged twenty-three years, had been subject to well-marked epilepsy of the convulsive form from the age of seven years, when he received a severe wound upon the head from the arm of a windmill: a marked cicatrix is yet visible. He was brought before a jury on two occasions upon a charge of burglary. On his second

examination he was seized in open court with a well-marked convulsion. His mind had become much weakened, so much so that he was considered imbecile. He was released on both occasions from custody, much to the dismay of the magistrates and neighbors. The former, a few weeks later, united in a petition for his sequestration in an asylum, which was granted: in this way he came under the care of Dr. Kelp. From the date of his admission, June 10, 1870, to that of the report, January, 1872, he had but six mild attacks, and these occurred during the first six weeks. His intelligence, memory, expression, and manners have all improved remarkably since his admission; yet, Dr. Kelp adds, "I should hesitate to pronounce upon his irresponsibility, in spite of his greatly improved condition, so fully am I persuaded that, should he be allowed to return home and to resume his former vagabond life, the epileptic convulsions would reappear, and would exert the same injurious influence upon his mental powers as they had previously done."*

That the presumption of moral irresponsibility is in favor of the epileptic accused of crime may be fairly concluded from the weight of authority, from the exercise of the reason upon the facts I have given to you, and from a fair application of the rule that the accused is entitled to the benefit of any reasonable doubt of his responsibility and culpability.

The following remarkable passage by Ray (Trial of Winnemore, "American Journal of Insanity," October, 1867) shows that the venerable Nestor of American psychology arrived at this conclusion nearly a decade ago: "In view of what we already know of epilepsy, and what still remains to be learned, we have a right to require the utmost circumspection and the closest investigation whenever the legal liabilities of epileptics are in question. The fact of its existence being established, is it going too far to say that legal responsibility is presumptively annulled, and that the burden of proof lies on the party that alleges the contrary? People are scarcely ready for it yet, perhaps; but to that complexion will they come at last."

In Germany, if we may judge from the writings of J. B. Friedreich, of Bavaria, they have "come to that complexion"

* Psychological Journal, vol. vi., Oct. 1872.

already. He says, "Criminal responsibility is absent in epileptics, even should it be proved that the determination to commit a criminal action resulted from revenge or malignity."

It was my opinion at the time of Klingler's trial, and further study of the subject of epilepsy has but served to confirm me in my conclusion, that, the grand fact of epilepsy being proven, he was entitled to a verdict of acquittal at the hands of the jury, on the ground of a most reasonable doubt as to his criminal responsibility.

In conclusion, regarding the disposal of cases of this character,—of persons acquitted of crime on the ground of insanity, whether of epileptic origin or not,—I would state that while I would extenuate his faults and seek to measure the full allowance of justice and of mercy to the prisoner, I would not forget what is due to the community at large. The homicidal lunatic should not be liberated upon an outraged community, seeking its own self-preservation and asserting its claims to see the laws executed, which alone can protect life, secure property, and preserve the rights and liberties of individuals. On the contrary, I maintain that when an individual's life has been saved by the plea of insanity, his sentence should be confinement in an insane asylum for life, where his dangerous and destructive propensities can be held in check and the public be made secure from alarm or injury. It matters not that he may be sane when acquitted, "and that a sane man should not be incarcerated in a lunatic asylum;" "*aux grands maux les grands remèdes*;" and no person who has been proved liable to explosive fits of homicidal insanity should be allowed his liberty because of an apparent convalescence, the continuance of which no expert, no matter how great his attainments and experience may be, can guarantee to the public. Nor, on the other hand, is it just or humane that he should lose his life because, although not responsible for a homicide actually committed, he might repeat its perpetration.

LECTURE XXII.

CHOREA.

Uncertainty of its Pathology.—Causes.—Complications with other Diseases.—Symptoms.—Treatment.

GENTLEMEN,—We have now come to the consideration of an affection upon which I shall not dwell at length, and the pathology of which is not determined. I refer to chorea (from the Greek *χορεία*, a dance), commonly known as St. Vitus's dance. In this disease there appears to be "an insanity of the muscles," our attention being first attracted by ludicrous, tremulous movements, by peculiar involuntary contractions and relaxations of those muscles heretofore under the control of the will. Hence there can be no doubt that those parts of the brain which preside over volition, the "centres for movements," are most seriously implicated.

The total absence of anatomical changes in the majority of cases, on post-mortem inspection, makes the pathology of chorea very uncertain. In fact, it remains a question *sub judice*, requiring further investigation. There is much difference in the views entertained by various observers as to its pathology; some claiming that there is simply a perversion of nervous function, the result of impaired nutrition. Some trouble in the motor centres exists, undoubtedly; the power of co-ordination is disturbed, and the mental faculties become involved, probably from the anæmic condition extending from the central portion of the brain towards its periphery. Hughlings Jackson claims that the disease is due to a rheumatic condition of the blood, producing a deposition of fibrin upon the cardiac valves. Small particles of this deposit are washed away by the blood-current and lodged in the minute vessels supplying the motor tract, especially the *corpora striata*, producing an anæmic condition of those portions of the brain, and, consequently, a hyperæsthetic state of the nerve-centres.

Post-mortem section has, in some cases, revealed the presence of small emboli in the regions specified: yet I doubt whether this theory deserves the importance which its author claims for it, and am rather inclined to regard the phenomena as consequences, rather than causes, of the disease.

Among the most prominent predisposing causes of chorea may be mentioned age, sex, and hereditary tendency.

This disease has been observed in infants, in adults, and in old people; but the vast majority of cases occur during the period between the eruption of the permanent teeth and the full establishment of the changes inaugurated at puberty. Before the second dentition, and after twenty years of age, cases of chorea are rare, and this infrequency increases in proportion to the length of the interval between a given age and the periods mentioned.

Carefully compiled statistics show that females are much more liable to this disease than males,—nearly in the proportion of five to two.

I have had occasion to refer often to the transmutation of nervous diseases, and the strong influence of an ancestral taint in the production of the neuroses in descendants. The truth of these propositions is very evident in relation to chorea. There are cases upon record of a child born of a choreic mother being afflicted with the same disease from the moment of its birth; and a considerable number of choreic patients have chorea in the family history, either in their parents or in the collateral branches of the family tree. Where chorea itself cannot be traced, you will, in most cases, find the history of insanity, epilepsy, neuralgia, hysteria, asthma, or habitual drunkenness in the ancestry.

Among the exciting causes, rheumatism holds the first rank. It is certainly often associated with the rheumatic diathesis, and appears to be one of its manifestations. This fact causes experienced physicians—those who are familiar with the natural history of chorea—to auscultate the heart with the same care that they would observe in acute articular rheumatism. Knowing the close relationship that exists between the two affections, you can frequently predict the probable development of rheumatic attacks, of an acute or subacute character, after your patient has recovered from chorea. Trousseau drew attention to the fact that chorea frequently appears among the sequelæ of scarlatina, and explains

its occurrence by the fact that a morbid condition similar to rheumatism, if not identical with it, is one of the consequences of scarlatina: thus chorea, as well as pericarditis, endocarditis, and pleuritis, following this exanthem, is most plausibly explained.

I once treated a young girl for chorea, who afterwards died of phthisis pulmonalis. Every member of her family was remarkably rheumatic; and I remember that, on auscultating her chest, I heard a systolic murmur in the neighborhood of the mitral valve.

There is no doubt that chorea sometimes precedes rheumatism, and that, *vice versa*, rheumatic attacks often lead to chorea. These facts will never be overlooked by the well-informed physician.

The etiology of chorea in other cases is often obscure. The moral causes (as in epilepsy), such as fright, anger, etc., are probably to be counted among the most common; dentition, menstrual derangements, with chlorosis, intestinal irritation, *onanism*, and last, but not least, gestation, are among the most common exciting causes.

It is not very uncommon to find chorea complicating other diseases, such as hysteria, hemiplegia from cerebral hemorrhage, and embolism. In the latter class of cases it is limited to the paralyzed side: hence the term hemichorea.

The symptoms of chorea are such as are not likely to be confounded with those of any other disease. Before the peculiar muscular movements appear there are certain prodromic symptoms, which will be observed, if not by the physician, by the mother, when the patient is a child. A certain mental change manifests itself by loss of interest in ordinary occupations or amusements; peevishness or irritability of temper, a mental hebetude, and impairment of the memory are among the mental phenomena. Restlessness, insomnia, and loss of appetite may also be noticed.

The muscles of expression, and those of one of the extremities (generally one arm), are first affected with painless, clonic spasms, which are entirely involuntary. The patient continually makes peculiar involuntary grimaces, and contortions of the face, which give it a comical expression. If you ask him to show his tongue, it will not be protruded calmly or evenly, but it is suddenly thrust forward in a very remarkable manner, and, after a short pause, it is as suddenly retracted. This characteristic, sudden protrusion of the tongue, termed the "choreic thrust," is almost pathog-

nomonic of the disease; although cases occur without its presence. Chorea is generally more manifest in the face than elsewhere; but it almost invariably extends to the extremities, and causes all sorts of muscular contractions. Continual jactitation is developed; the patient is unable to remain quiet an instant, neither can he perform those finer movements which require muscular precision, such as picking up a pin, tying a cravat, etc. He is in a condition of continual nervous restlessness, which only temporarily ceases during sleep. In exceptionally bad cases this involuntary muscular spasm is continuous even during sleep; and unless rest is induced, and the irritation allayed by remedial measures, the patient becomes exhausted, and a fatal result ensues. But, fortunately, in most cases the duration of the disease is short, and its tendency is towards recovery.

Sometimes choreic manifestations are developed during gestation. The affection under these circumstances is occasionally of a most inveterate and desperate character, and it may then become a source of great anxiety to the physician. The question of inducing abortion, or premature labor, may arise, when the continuance of the muscular agitation threatens the life of the woman and the foetus has reached a viable period. Anæmia frequently complicates chorea: in fact, it seems to be one of its concomitants.

Often, as the disease progresses, especially if the muscular agitation is extreme and such as to occasion alarm regarding the termination, the mental symptoms of the prodromic stage increase in gravity. This occurs because the upper portions of the brain are involved in the morbid processes. The lower portions, such as the corpora striata, are always affected from the beginning; but it is probable that the hemispherical cerebral masses become more or less anæmic as the disease progresses, and, as a consequence, the cortical cells of the supreme cerebral centres no longer work harmoniously. Their implication is evinced by the moral change, great irritability, moroseness, peevishness, sleeplessness, increasing impairment of the mental faculties, and a characteristic hebetude of countenance. Delirium of a low, muttering character may supervene upon the other symptoms termed typhoid or adynamic. These symptoms are truly alarming, especially if the disease continue longer than usual.

The specific gravity of the urine is very decidedly augmented : this is due to the increased formation of urea, dependent upon the excessive retrograde tissue-metamorphosis caused by the continual muscular movements. The increase of urea and also of phosphates and lithates in the urine is what makes it heavier. When its specific gravity diminishes, the return of health commences, and the end of the disease is near.

Another fact which I wish to impress upon your minds is, that in chorea the digestive functions are more or less impaired, and the disease is of less severity in proportion to the toning up of these organs.

This brings us to the consideration of the *treatment* of chorea. In this disease there seems to be a definite duration, a self-limitation, of the morbid processes: this is generally of remarkable regularity, about four or five weeks. Trousseau has little faith in therapeutic measures, except as means of palliation, probably because there is a natural tendency towards recovery. Therefore you understand why so many nostrums are in repute as being able to effect its cure. The argument *post hoc ergo propter hoc*, in medicine, has caused the constant postulation of the virtues of so many drugs in the treatment of this and other self-limited diseases. You will find physicians varying in their therapeutic experience, frequently deriving opposite results from the same remedy given in the same disease and under apparently the same circumstances. Thus, some rely upon veratrum viride for the cure of fever, others upon digitalis or quinine, when, in fact, too many overlook altogether that *vis medicatrix naturæ* which accomplishes more than all medicines combined. Diseases which from their very nature abate after a definite period are often considered cured by the last medicine administered, which thus receives undeserved credit. Hence you appreciate the great amount of contradictory evidence brought forward to substantiate the value of medicaments, and the reason of the formation of diametrically opposite conclusions from facts of experience.

There are several considerations which I desire to impress upon your minds in connection with the treatment of chorea. The first is only a reiteration of what I said in relation to epilepsy; namely, take care to place the patient under the best possible hygienic conditions, give tonics, assist digestion, and counteract insomnia.

Shower-baths, if the season permits, are very good, and relieve the severity of the disease. Next to these, occasional warm sulphur-baths, when judiciously employed, the patient being guarded from taking cold, often give the happiest results. They act by alleviating the nervous erethism and producing sleep, as well as by the good effect exerted upon the cutaneous surface.

Then the administration of some good ferruginous preparation, such as the citrate of iron, the tincture of the chloride of iron, and other similar compounds, with small doses of quinine as an adjuvant, is always beneficial.

The preparations of zinc and arsenic have great reputation in the treatment of chorea. By some arsenic is considered almost a specific. When I give it I generally use Fowler's solution in combination with iron, always being careful not to give too much of the arsenic, which would derange the gastric functions. In the use of arsenic, I advise you to give it invariably after meals, never upon an empty stomach. I believe that arsenic does possess a certain specific influence in the palliation of chorea. C. B. Radcliffe has employed Fowler's solution by hypodermic injection, and claims to have found great benefit from its use in this manner. He advises its dilution with distilled water, one part of Fowler's solution to three parts of water. No bad results followed its use in this manner, either locally or generally, while the chorea rapidly diminished in intensity and its duration was apparently cut short to a considerable extent.

Strychnia enjoys a high reputation with many practitioners of large experience. It is to be given in doses rapidly increasing from the minimum until the physiological effects are manifested; the dose is then diminished for a few days, and afterwards again increased. It acts, probably, by invigorating the digestive organs, as well as by causing a determination of blood towards the nervous centres.

Radcliffe highly recommends the free administration of alcoholic stimulants in these cases. He says that large quantities are not only tolerated, but received with benefit, by the unhappy subjects of chorea.

The systematic application of electricity to the principal nerve-centres, either by the faradic or the constant current, is probably productive of some good effect.

As regards gymnastic exercises and the Swedish movement cure, I make no comment, as I believe their value to be greatly exaggerated. However, gymnastic exercises under the direction of a competent teacher would probably be of advantage in the slighter cases, and in the more severe ones when recovery has advanced to some degree. In stammering there is something like a chorea of the muscles of articulation, and great benefit is derived from a systematic training of the rebellious vocal apparatus.

Another method of treatment recommended by high authority is the use of ether spray to the spine. This consists in the diffusion, by means of an atomizer, of a certain quantity of ether over the spinal column about twice a day; and it is said to have been extraordinarily successful in some cases. This may be the case; but I have been so often disappointed by new remedies that I advise you not to be too sanguine in their adoption, but to accept them with a certain prudent reserve. Enthusiasts are oftentimes carried away by the last remedy which they may have used with success, and then, for awhile, will anticipate results from it which they will not realize. You will also find that the use of therapeutic measures is too often dependent upon fashion, which sometimes pervades the medical world: when a new means of cure is announced to the profession, some practitioners greatly exaggerate its efficacy and throw everything else aside.

To come back to chorea and its treatment, conium strikes us as being probably efficient, because, as I told you when speaking of insanity, it has a positive influence over the motor nervous system. In chorea, the motor nerve-centres of the brain are preternaturally excited; from this we might suppose that the quieting influence of conium ought to be remarkable in the treatment of this affection. It is, however, far from being a specific.

The inhalation of chloroform and ether has been loudly praised by some, and as loudly denounced by others. I have never witnessed any permanent benefit from this use of these drugs in chorea, and, theoretically, can see nothing in their probable mode of action and known effects to warrant further experiments with them.

I will speak to you of one more remedy which sometimes gives remarkable results, but which is not sufficiently appreciated by practitioners, in the treatment of chorea. This is the hydrate of

chloral. If there is persistent insomnia, there is nothing equal to it in procuring the necessary sleep. It is a hypnotic, not an anodyne (like opium), and one that can be safely administered, provided it is given with due caution. In the chorea of pregnancy, where the nervous agitation is extreme and generally accompanied by insomnia, chloral is the best medicine that can be administered; and, independently of its hypnotic powers, it is one of the best antispasmodics that we possess.

If you will look over the recent literature of tetanus, you will find that chloral ranks very high as a remedy in the treatment of this terrible malady, because of its relaxing effects upon the spastic muscular spasms; and it probably exercises an equally beneficial influence over the clonic as over the tonic muscular contractions.

Of the use of nitrite of amyl in this affection I have no experience, nor have I seen any reference in the journals to its administration by others: it seems to me that it is hardly indicated, in consequence of the persistency of the clonic choreic spasms,—the effects of the last-named remedy seeming to be most beneficial when it is given during an appreciable interval in the convulsions over which it has manifested such wonderful control.

LECTURE XXIII.

CHOREIC AND EPILEPTIC HEMIPLEGIA.—HYSTERICAL, RHEUMATIC, AND LEAD PALSY.

Choreic Hemiplegia.—Hysterical Paralysis.—Globus Hystericus.—Differential Diagnosis.—Rheumatic Paralysis.—Lead Palsy.—“Wrist-drop.”—Treatment.

GENTLEMEN,—In my last lecture I spoke of the characteristic features of chorea ; all of which, I hope, have been sufficiently impressed upon your minds. I omitted, however, to dwell upon the fact that the choreic movements are frequently more marked on one side of the body than on the other,—sometimes, indeed, are altogether confined to one side,—and that hence there is a certain coincidence in this respect between chorea and epilepsy, in which the convulsive affection of the nervous system sometimes produces a greater muscular agitation on one side of the body than on the other, leading to a greater consequent exhaustion both of the nervous centres controlling the muscular movements and of the muscles themselves on the side most affected. Following *chorea*, as well as *epilepsy*, we exceptionally find an attack of a hemiplegic character. I have repeatedly told you that when hemiplegia follows epilepsy the peculiarity of the paralysis is its extreme transiency, lasting sometimes but a few hours, and at most two or three days ; and that in persons thus affected it is prone to manifest itself again after the next violent paroxysm ; also, that the hemiplegia is most likely to occur on the side which has been most severely convulsed, although it is sometimes developed when both sides have been equally affected. When hemiplegia follows chorea, it also affects the side that most severely suffered during the latter state. But, though of shorter duration than hemiplegia following some brain-trouble, the choreic variety lasts longer than does that dependent upon epilepsy. The hemiplegia of chorea may be of several months' duration. But, gentlemen, there is seldom any difficulty about the diagnosis of choreic hemiplegia, and it is not very liable to lead you into error. Its

tendency is always favorable, the disease intrinsically leading to recovery.

Again, remember that in choreic hemiplegia there is no evidence whatever of an involvement of the cerebral centres, or of a brain-lesion of any kind, as is the case when a clot is in the brain,—the phenomena of which we have already carefully considered. Whenever certain brain-affections lead to hemiplegia, we almost always find a paralysis of the tongue, of the palate, of the face, and of the muscles of articulation. In choreic hemiplegia the paralysis exists on the side of the body which has been most agitated by convulsions, but there is never any evidence of facial palsy or of paralysis of the muscles of the tongue, neither is there a very persistent degree of mental impairment,—all these being symptoms of brain-lesion: thus we are enabled to make a correct diagnosis. The choreic paralysis is also of a much less profound and complicated character than that resulting from a cerebral trouble; but a remarkable symptom, and one which surely ought to prevent you from being led astray, is the peculiar manner of protruding the tongue, the “choreic thrust,” which I described in my last lecture, and which should enable you to diagnosticate correctly. I do not wish you to understand me as intimating that chorea and epilepsy are always followed by hemiplegia; on the contrary, the supervention of hemiplegia in both diseases is generally the exception; but I want you all to be on your guard, so that should it appear, you may not be taken by surprise. The etiology of the affection might be explained by the atonic condition of the nervous system, resulting in complete neurasthenia, attributable to the prolonged disturbances. Should, exceptionally, an attack of hemiplegia supplant one of choreic convulsions, it does not in the least necessitate a change of treatment,—this condition being amenable to the same therapeutic measures as those indicated in the management of uncomplicated chorea, such as tonics, some ferruginous and arsenical preparations, and the judicious use of the faradic current on the hemiplegic side, and of the continuous current upon the spinal column.

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annoy the physician and alarm the family ; and where it exists, however slight it may be, should you be unable to recognize its character, you necessarily will fail to relieve the unreasonable apprehensions its presence excites ; while, on the other hand, should you erroneously diagnosticate a brain-trouble, your prognosis from the supposed origin must necessarily be grave and guarded. In all forms of hysterical paralysis the prognosis is favorable, because, unlike many other varieties, it does not depend upon any organic change of the nervous elements, but is generally the result of some perverted condition of nutrition of the nervous centres, giving rise to characteristic phenomena, which, by judicious treatment, can easily be relieved. Now, what forms does hysterical palsy assume? What are the *diagnostic marks* that indicate it and enable us to recognize it? Hysterical paralysis, as well as choreic paralysis, is marked by the peculiar *superficiality* of the paralytic phenomena, which latter is not profound,—not complicated, as in those forms that depend for their causation upon cerebral lesions. Hence it is incomplete ; oftentimes partial and limited ; it may affect one muscle, or a joint, or a group of muscles, and sometimes assume the paraplegic, but very rarely the hemiplegic, form. This last form, though more common in chorea and in epilepsy, is really the exception in hysteria ; in spite of which it may occur, and cause you some embarrassment in arriving at a proper diagnosis. However, I shall endeavor to familiarize you with its protean forms, so that whenever you encounter it you will rarely experience any difficulty in distinguishing all its features. To women of a hysterical character authors generally ascribe certain peculiarities of expression, such as a remarkable fullness of the upper lip, a certain drooping of the upper eyelid, and a singular smoothness or softness of the surface of the body. But if you rely too much upon these appearances, you may expect to be led into error. There is always, it is true, a peculiar and undue mobility of the emotional centres, leading to disturbances of their equilibrium, by which hysterical women, and also hysterical men, are characterized. They are prone to an excitation of the emotional faculties, causing them to laugh and to cry with great facility, the motive thereof not being apparent.

But a peculiarity with which the experienced practitioner is well acquainted—and which you will better appreciate when we

come to the study of hysteria proper—is the tendency to the sensation called “globus hystericus,” which is an imaginary perception of a ball rising from the hypogastric region up to the throat, and producing a distressing attack of suffocation, followed by repeated attempts at deglutition. Then another accompaniment is what is known as the *clavus hystericus*, when the patient feels a pain in the cranium, generally in the frontal region, resembling the sensation which would be produced by the driving of a nail into the head. Again, there is a tendency to void enormous quantities of limpid urine, a characteristic of hysteria which is suggestive of the influence exerted by the emotional disturbances upon the vaso-motor nerves of the renal vessels, causing sudden hyperæmic conditions of the kidneys, vascular distention, relieved by an increased flow of urine. Whether this explanation be correct or not is a question *sub judice*; but that the fact exists there is no doubt whatever. Should you have a female patient with symptoms of paralytic phenomena, and should you be in doubt as to whether these are of hysterical origin or not, the simple absence of the evidences of a hysteric constitution will be no proof of the non-existence of hysteria; but their presence will be an additional confirmation of the existence of the hysterical nature of a given case of paralysis, provided the latter behave as hysterical palsy generally does. The hysterical palsy is almost always limited or restricted in character; not infrequently the affection is confined to the larynx, giving rise to hysterical aphonia. This is recognized by the fact of the sudden and causeless appearance of the paralytic phenomena, and their equally sudden and causeless disappearance, which latter condition is almost pathognomonic of all the varieties of hysterical paralysis. If, in a given case of hysterical constitution, a woman goes to bed at night without any ailment whatever and wakes up the following morning voiceless, you immediately examine her in order to ascertain if the aphonia is due to some catarrhal affection of the larynx; but the mucous membrane is found healthy, there are no growths, no catarrhal hyperæmia, and the laryngoscope fails to reveal any pathological condition; a fact, in connection with the suddenness of the advent of the attack, fully warranting you in diagnosing hysterical aphonia, and in predicting a sudden and unexpected disappearance of the trouble, corre-

sponding with the abruptness of its appearance. Whenever there exists a paralytic condition of certain muscles, produced by a pathological state of the nerve-centres, just in proportion to the want of use of these muscles will there be a corresponding atrophy, which, if not prevented or arrested, will end in a fatty degeneration of the ultimate muscular fasciculi. But in hysterical palsy this is not the case; and an important feature thereof is that there never is any evidence of atrophy, the rotundity and size of the limb being perfectly preserved, even where the muscles have not been used for a long period. Again, if the patient has hysterical palsy, the facial muscles are never involved, and you can lay it down as a rule to which there are no exceptions, that in hemiplegia the result of cerebral disturbances there is invariably more or less facial palsy, while in the hysterical forms of paralysis, even when hemiplegic in character, there is never any paralysis of the velum palati, of the tongue, of the buccinator, or of the facial muscles. This feature alone is sufficient to lead to a correct diagnosis; and should this facial palsy exist, you can readily exclude hemiplegia of a hysterical type. And, as averred by Dr. Todd, the hysterical hemiplegia can always be positively diagnosticated, partly by the symptoms just alluded to, and also by the peculiar and *characteristic walk*. If you ever have noticed an individual affected with hemiplegia the result of brain-trouble, you will have observed that the entire weight of the body is thrown upon the limb of the non-affected side, and that the affected limb is not brought in advance in a straight line, but that the foot makes a sweeping movement, describing an arc of a circle. This walk, characteristic of cerebral hemiplegia, is not met with in hysterical hemiplegia; the patient rests upon the non-affected limb, but in walking drags the paralyzed limb forward in a straight line, like an inanimate mass. With all these characteristic differences before you, I think it would be scarcely possible for you to arrive at erroneous conclusions. You have first the phenomena of the hysterical constitution,—the evidences of a hysterical diathesis, so to speak: these sound the note of alarm; though by these alone you may be misled. But you have also the superficiality and the abrupt advent of the paralytic phenomena, with the corresponding rapidity of their disappearance. In the phenomena characterizing all forms of hysterical palsy there is

a remarkable incompleteness and superficiality, something wanting, which, if you are careful, will not escape your observation.

Then there may have been a previous attack of hysterical aphonia; besides this, do not forget to attach great importance to the fact of the preservation of the size and rotundity of the limb affected, and the non-impairment of the muscular electro-contraction. Again, there is no facial palsy; an absence of all brain-symptoms; no evidence of mental impairment; and the peculiar manner of walking. These symptoms are almost pathognomonic, and should enable you to recognize this disease without additional description.

I will give you an illustration taken from my own experience. Years ago I attended a young lady who, whilst in perfect health, in getting up from her chair had been suddenly seized with symptoms of a paralytic character. The friends, greatly alarmed, sent immediately for me. Upon inquiry, I could not obtain the slightest evidence of a hysterical constitution. But one fact exciting my suspicion was that the patient was about sixteen years of age, and her menstrual functions were not quite established, which was a rather tardy development. Recollect that I have often cautioned you to be on your guard as far as the coincidence between neuroses and pelvic troubles in women is concerned. This young lady could neither rise from her chair nor walk, even when supported by two strong men. Not feeling certain of the nature of this paraplegia, I sought to ascertain in what manner it was produced. I excluded spinal troubles. There was evidently no hemorrhage in the spinal cord, which might lead to sudden paraplegia. The health of the patient was favorable; cheeks rosy, and appetite good; there had been no gradual invasion of symptoms; she had lifted no weight, had received no injury, had made no unusual efforts of any kind. After careful examination, having excluded organic disease of the cord, I began to suspect that she was hysterical. Hysterical phenomena are not merely simulated, as many physicians suppose; they are doubtless exaggerated, but they are not assumed; hysteria is as much a disease of the nervous system as is chorea or epilepsy. I diagnosticated hysterical paraplegia, and so told the parents; gave tonics,—iron, quinine, and strychnia (which increase the amount of blood in the spinal cord, as opposed to ergot and bella-

donna, which diminish it),—and systematically used electricity. The patient eventually recovered, the paraplegia having lasted about six months; and a noteworthy feature was that the suspension of muscular action during that length of time was unattended by the least wasting away of the muscles of the lower limbs, which remained as rotund and perfect as before the attack. In paraplegia from serious causes electricity is employed to prevent atrophy and subsequent fatty degeneration: in this case, however, it was only used as a general nerve-tonic. Another peculiarity which I must mention is the patient's recovery, which was as rapid as the seizure was sudden. In this case the voiding of an abnormal amount of limpid urine, the globus and clonus hysterici, were absent, but there was a quasi-hysterical look, and, as the French term it, a "*je ne sais quoi*," which was characteristic, and she will remain more or less hysterical during life.

Suppose there had really been an inflammatory or hyperæmic condition of the spinal cord: would it have been advisable to give strychnia? Certainly not; for strychnia increases the amount of blood in the spinal cord; which property makes it quite valuable in those forms of spinal trouble which are due to an anæmic condition of the cord; and it is possible that the common pathological condition of hysteria, of chorea, and of some forms of epilepsy is an anæmic state of certain nervous centres, producing exhaustion and depression: the treatment, therefore, should be directed towards the production of an afflux of blood to these centres.

There may be, as observed by Todd, certain varying conditions of polarity, corresponding with which are accompanying symptoms of irritation and of depression,—these last being manifested by paresis or paralytic phenomena.

This state evidently calls for such tonics as iron; but never give alcoholic stimulants in chronic complaints, especially when of a hysterical character; the physician who is imprudent enough to administer them in such cases is culpable, and responsible for moral consequences oftentimes of a disastrous nature. So much for choreic, epileptic, and hysterical hemiplegia.

Paralysis sometimes manifests itself in connection with the *rheumatic diathesis*. Whenever this latter affection exists, there is a poisoned state of the blood; and, as a result, the muscular

tissues become poisoned, and certain groups of muscles are generally affected. The rapidity of the development of this form of palsy is one of its characteristic features. All muscular movements of the affected parts are very *painful*; which, taken in connection with the enjoyment of full intellectual vigor, will obviate all diagnostic difficulties. I have told you how to recognize hysterical hemiplegia, partly by the absence of brain-symptoms, there being no manifestations of cerebral trouble. Rheumatic paralysis is also, as just stated, marked by an absence of head-symptoms; and it can be recognized by the fact that it is never hemiplegic or paraplegic, but is always limited in character, seldom affecting more than one muscle or group of muscles. The peculiarity of this palsy is that the slightest movement of the affected muscles is attended by violent pain; therefore these patients are very particular to remain as quiet as possible.

I shall conclude by speaking of a very important form of palsy, one which you may be frequently called upon to treat, and which you should always be able to recognize. I refer to lead palsy. This paralysis is peculiar to persons who work in lead,—painters, plumbers, etc. I shall present a grouping of the prominent clinical features of the disease, in order to familiarize you with them. There is the presence of a certain constitutional state,—a lead diathesis, if I may be permitted to use the expression,—an evidence of which we obtain first from the history of the case. The patient is usually a painter, and is subject to attacks of colic, sometimes very violent, known as *colica pictonum*, or painter's colic, which is generally attended by obstinate constipation. Such colic occurring, and knowing the avocation of the patient, you at once suspect lead-poisoning. You examine the gums, which in the latter condition show a characteristic blue line, indicative of the deposition of lead. Should a tooth be missing, however, there will be an interruption of the line corresponding with the vacancy; and this is a feature of the lead line.

How does the lead get into the system? It has three different means of entrance. The *first* is by the saliva. Some painters are careless, and will sometimes, in chewing tobacco, introduce it into the mouth with the hand covered with lead paint. The *second* is by inhaling impalpable minute particles of lead floating in the air and absorbed by respiration. The *third* way is by absorption

through the skin. You all know that the cutaneous surface possesses very decided absorptive power, of which you have probably been convinced from the peculiar garlicky odor emanating from the breath of a person to the soles of whose feet onion-poultices have been applied. The history of the case, the characteristic blue line, the *colica pictonum*, and the obstinate constipation will enable us to arrive at the proper conclusion. Besides these is the phenomenon known as "wrist-drop," which is a paralysis of the extensor muscles of the hand, and of the muscles of the ball of the thumb. These are the muscles mostly used by painters.

One peculiarity in this disease is, that the first evidence of the affection of the nervous system is peripheral, and not central, in character. The effects of the poison are first manifested in the extremities, and if not subjected to therapeutic measures it soon becomes centripetal in tendency, when the biceps, triceps, deltoid, and other muscles become involved, the affection finally traveling to the brain, in which organ the lead can be detected in a post-mortem analysis. In this case there will be wasting of the convolutions, and sometimes softening, and a depression and widening of the sulci. Epileptic convulsions during the last stages will be symptomatic of the implication of the nerve-centres. The next questions which you might properly ask are, Why are the extensor muscles singled out by the morbid processes? and, Why is the paralysis peripheral in character? Because the lead is most rapidly absorbed by those parts requiring increased nutrition. The greatest flow of blood is evidently to those parts that are most used and whose functional activity is greatest. In these, the retrograde metamorphosis of tissue is very active, and the nutritive functions are correspondingly active, as is plainly evidenced by the hypertrophied arms of the blacksmith and the legs of the ballet-dancer. The disturbances or lesions it produces are not, at the outset, of an organic nature, at least so far as an involvement of the great nerve-centres is concerned. The muscles and nerves are probably first affected, and later in the complaint the great centres may become involved, unless the morbid process be arrested. The local symptoms of paralysis always precede the graver manifestations of the later periods of the disease. Epileptic convulsions, and even brain-softening, which appear late in aggravated cases as decided evidences of centric disturbances and changes, are invariably pre-

ceded by milder symptoms, such as the characteristic "wrist-drop," or paralysis of the extensor muscles. In what part of the body do painters manifest the greatest activity? In the thumb, the hand, and the extensors of the arm: these parts, consequently, receive the greatest supply of blood, which is surcharged with the poison. The flow of blood to them being more rapid, the poison must there first show its effects, causing the palsy to be peripheral. The mortality of the disease seems to bear a direct relation to habits of intemperance and long-continued exposure to sources of infection.

Now comes the question of treatment. There are few diseases in which relief can better be given, and where a proper method proves more satisfactory, than in lead palsy. The first thing to look to is the hygienic condition, which should be made as favorable as possible. The patient should be immediately removed from the noxious influences of his avocation. You cannot think of performing a cure if you allow as much poison to be absorbed as you are eliminating. This elimination can best be attained by a remedy which in this case is almost a specific: it is the *iodide of potassium*. This is really as great a specific in lead-poisoning as quinia is in malarial poisoning, and, according to Melsens, the iodide of potassium is a specific in all mineral poisons deposited in the tissues. This remedy, by an elective affinity, seeks the lead, which can readily be eliminated by the excrementitious channels. This is also the case in mercurial poisoning, which often occurs in the treatment of secondary syphilis. In these cases you frequently find people whose systems are so surcharged with mercury that they have as much mercury as syphilis. By treating them with iodide of potassium you may salivate them without giving a single additional grain of mercury, which latter is re-absorbed, and as a consequence you pyralize or re-mercurialize your patient. Therefore be cautious in its use in mercurial poisoning. I only cite this fact as an example of the eliminating power of iodide of potassium.

As a *prophylactic* against lead-poisoning, *sulphuric acid* is most efficient. This acid combines with the lead, forming the sulphate of lead,—an insoluble salt, and one not readily absorbed. It is employed to a great extent in lead-factories, and is given as a prophylactic in the form of sulphuric-acid lemonade, of which

the workmen freely partake, effectually preventing lead-poisoning in most instances. In fact, there is nothing better to prevent toxæmia from saturnine sources. *Sulphur baths* are also very efficient in the treatment, and may be freely administered. One more fact in regard to the treatment of lead palsy is that excellent effects are to be obtained by combining *ferruginous preparations*, such as the citrate of iron and quinine, with the iodide of potassium. For where there exists a toxic condition of the blood from a mineral poison the tissues are ill nourished and anæmic; and nothing is better calculated to relieve this condition than the preparations of iron. The last method of which I have to speak is the judicious use of some form of *electricity*, especially faradization of the affected muscles. In lead palsy there is a diminution of contractility in the fibres of the affected muscles, which might ultimately lead to a fatty degeneration. Seek to avoid muscular atrophy, by employing mild faradic currents, and thereby preserve the original size and form of the affected limb and restore the tonicity of the muscles.

LECTURE XXIV.

ALCOHOLISM.

Alcoholism a Disease of the Nervous System.—Delirium Tremens.—Effects of Alcohol upon the System.—Oinomania.—Alcohol not entirely eliminated from the Body.—Causes of Alcoholism: Occasional Causes; Predisposing Causes.

GENTLEMEN,—To-night we shall discuss a subject replete with interest,—one which, as physicians, will frequently claim your earnest study, and will occupy your attention almost as constantly as any other of the common varieties of disease. I allude to alcoholism. Alcoholism is a disease of the nervous system. It is superinduced by a poisoned state of the blood, which, surcharged with alcoholic poison, occasions a morbid condition of the nervous structures. As drunkenness, with all its concomitant vices, degrades the human race and makes a beast of man, and as the propensity to this evil has ever been on the increase till it has become the great curse of the day, it is highly necessary for you to study all its peculiarities with great care,—not from a moral point of view, but as practical physicians, so that it may be in your power to render immense service to some of your fellow-beings, by rescuing them from a condition as unfortunate as it is lamentable. If you will carefully follow me to-night in the consideration of the nature and treatment of alcoholism, you will acquire a proper understanding of the disease, and be placed in possession of a vast amount of information which will undoubtedly be of invaluable assistance in your future labors. For the perusal of even many works upon this subject will oftentimes disappoint you by the paucity and barrenness of their contents. Therefore I shall dwell at length upon this disease, culling for your benefit the views of the best authorities, at the same time endeavoring to be as concise and clear as possible in giving you the results of my own experience.

As I have just stated, alcoholism is a disease of the nervous

system. It may be either acute or chronic. If it be chronic, as is generally the case, you will find certain prominent characteristics with which it is especially stamped, so that it cannot be very well mistaken for any other disease, even independently of the history of the case. Among these characteristics are the peculiar tremor, the disturbance of the motor centres, as evinced by the peculiar tremulous muscular motion, a morning vomiting, not unlike the morning sickness of pregnancy, insomnia, anorexia, and a great liability to hallucinations, especially of an auditory and visual character.

The acute form of alcoholism is sometimes called *delirium tremens*, or *mania a potu*, and may be confounded with acute alcoholic mania, or acute alcoholic insanity. The intensity and the manifestations of the different phenomena of alcoholism are very dissimilar in different persons. In some, for instance, alcoholism may exist during the greater part of life without any delirium tremens; in others, the delirium is the only phenomenon ever developed. Now, in chronic alcoholism the blood is continuously surcharged with the alcoholic poison, so as to constitute an alcoholic diathesis. This form has only recently been commented upon, and is of comparatively modern date. It has been most admirably described by Dr. Anstie, of London. The history of alcoholism is the history of alcoholic excesses,—of the reckless and immoderate use of alcohol as a beverage, without the exercise of self-control, and aggravated by the pernicious influences of the poor quality of the liquor, its degree of concentration, its deleterious foreign ingredients, and the frequency of the potations. Some persons, when taking a drink of whisky or of brandy, always dilute the liquor with a certain quantity of water, and in this way the pernicious effects are not so apt to be produced; but many, and those are the individuals who soon become chronic inebriates, “take their whisky straight;” and the frequent repetition of the use of such concentrated liquors is soon productive of the worst morbid phenomena. These are more particularly manifested after prolonged debauches or protracted sprees.

. The older writers dealt almost exclusively with the acute form of alcoholism,—the *mania a potu*,—and knew little, if anything, about the chronic form. The old views, of course, had a great influence upon the treatment; but our present knowledge of the

effect of alcohol upon the nervous system has rendered the primitive theories no longer tenable. By the older authors it was always maintained that delirium tremens was due to the withdrawal of the alcoholic stimulus; that it was in consequence of the suspension of the accustomed stimulant that the acute symptoms of delirium supervened. But Dr. Warren, of Boston, ascertained, by the study of upwards of one hundred cases, that delirium tremens nearly always proceeds from excessive debauches. It is after a protracted spree, from the excessive prostration of the nervous system thereby induced, that the nervous powers succumb and develop the condition of delirium tremens. There is, of course, a previous state of *malaise*, and of distaste for liquor; but this arises from the excessive abuse of alcoholic beverages; and the reason why the patient no longer indulges is simply because he is prevented by nausea; he is disgusted and unable to retain the liquor upon his stomach; he has the desire to drink, but the gastric irritability is too great, and causes him to vomit. This is only the commencement of the condition known as delirium tremens; it is not the cause, but merely the initiatory stage.

We have now developed the important fact, in opposition to the old theory, that delirium tremens is the result of alcoholic poisoning after prolonged debauches. Now we also know that, in overdoses, in large quantities, as it is often taken, alcohol is not stimulating at all; for through its continuous use the system is in such a condition of prostration and of exhaustion that stimulation by its means is an utter impossibility. In large and oft-repeated doses its action is as distinctly narcotic as that of chloroform, henbane, or opium, when given internally. Therefore, in small doses alcohol is a stimulant, in large doses it is a sedative. In drunkards, or chronic inebriates, with whom the use of alcohol has become a daily necessity, the system is in a state of constant depression, and no longer recoils. Hence in these cases alcohol has ceased to act as a stimulant. Again, in cases of alcoholism you almost invariably find that the acute manifestations are developed after a previous poisoned condition of the system has been intensified by new excesses. Therefore it is exceedingly rare to find delirium tremens in a man free from alcoholic poisoning; but it seems as if there were a certain limit to the powers of endurance of an individual, and that after the blood

has been surcharged with alcohol to a certain degree this will be manifested by an attack of delirium tremens. The system will tolerate only a certain amount of poison. This is not a mere theory, as you will see when I come to speak of the treatment of this disease; and I will say at once that if I have a patient entirely under my control, and he has been taking a daily average of several pints of whisky, I cut off his supply suddenly, and do not allow him another drop. Is it not absurd, after the system has been nearly saturated with poison, to introduce more of it? Does not every additional drop prove pernicious? Can it do any good to give more poison? Of course not: it can only aggravate the situation. I recollect a lady who was addicted to the use of morphia; she used to take thirty grains of it, in one dose, every morning. She was the daughter of a physician in the South, and was placed under my care for treatment. When asked how I intended to treat her, I replied that I purposed withholding the morphia at once. The remark was made that I would kill her by such a procedure; but I informed the father that I would assume the entire responsibility of my action; and, being a man of intelligence, he yielded, and I had his daughter under treatment for six months without giving her over five grains of morphia, or any other preparation of opium, in that time. Of course I had to substitute a mild nervine remedy for the morphia, in order to relieve the constant craving for the narcotic.

But I am an advocate of the total-abstinence system. If it becomes necessary to re-administer a narcotic, the patient can readily be placed back under its influence, which would not be the case should the withdrawal of an accustomed narcotic induce dangerous symptoms of depression; by a less energetic treatment, one blow after another would be added, and by continuing the administration of the pernicious substances craved, slight benefit would accrue from treatment, and it would become impossible to control the habit or to undo the mischief.

Let us see what are the effects of alcohol upon the system. If we dissect a nerve or a bundle of nerves, and then immerse it in strong alcohol, we shall find that after a certain period the nerve will no longer be a conductor of nerve-force, but will be absolutely paralyzed so far as the transmission of nervous impulse is concerned. But if we take only a weak alcoholic dilution, we

shall not find the effects so deleterious ; these paralytic phenomena will not become completely developed, and the transmission of nerve-power can still take place. Does not this clearly prove that the continued use of strong alcoholic beverages has a paralytic influence upon the nervous system ? Another very bad effect of alcohol is, that it prevents the proper oxidation of tissues in the body. Under its use there is an undue consumption of the oxygen of the blood, and instead of the tissues being well nourished by the oxygen carried by the red corpuscles, the hydrocarbon steals it and appropriates it for its own use. As a consequence of this defective tissue-oxidation, the blood becomes surcharged with fatty materials, and the deleterious effects of the excessive retrograde metamorphosis on the system soon begin to show themselves. Now, I repeat that the first condition, the *sine qua non*, of alcoholism is the existence of an improperly oxygenated arterial blood ; this blood does not furnish the necessary nutritive elements to the different organs, and their functional activity is impaired. Therefore it is that in drunkards laxity and flabbiness of tissue exist because of the excess of adipose structures, the mind grows heavy and sluggish, and if this condition is allowed to become aggravated, the degrading vice of drunkenness causes its victim to sink lower and lower in the physical, social, and moral scale.

Alcohol also manifests a wonderful effect upon the ganglia of the sympathetic system, as I shall soon explain. I have already stated that one of the effects of alcohol on the system is stimulating, and another sedative or depressing. Now, suppose any of you gentlemen, in a social manner, to take a glass of good sherry wine,—not too much, but a moderate quantity : what will be the result ? You will feel a genial warmth diffused all through the body, there will be an exhilaration of the animal spirits, an exaltation of the mental faculties, the entire system will be stimulated, the face will radiate a healthful glow, there will be an accelerated circulation of the arterial blood, and you will be in the condition of a man under a charming and pleasurable influence. But if instead of taking one glass of sherry you take several in rapid succession, or a goblet of whisky, what will happen then ? The large amount of alcohol, coursing first through the portal circulation, will soon produce its poisonous

effects ; there will be no longer a stimulation, but on the contrary a depression ; you will become incoherent in thought and in speech, the hypoglossal nerve will be affected, the tongue will be thick and move with difficulty, the walk will be unsteady, revealing the want of muscular co-ordination ; the action of the alcohol on the cerebral centres will depress the moral faculties, and you will use obscene language, of which you will afterwards be ashamed, should it come to your knowledge ; in short, you will become stupid, sleepy, and morose ; and should this condition be frequently renewed or incautiously prolonged, a true alcoholic coma, as I have described it in a former lecture, will supervene, and you may die from blood-poisoning. A sedative dose of alcohol affects the ganglionic centres of the sympathetic system ; the eyes become turgid with blood, and the face is flushed and congested,—conditions which are the tell-tale sign of this over-indulgence, due to a paralysis of the vaso-motor nerves, allowing relaxation of the coats of the arteries, and producing a state of hyperæmia. This shows the influence of the immoderate use of alcohol upon the great sympathetic system.

Now, gentlemen, many men become chronic inebriates without being aware of it, or even suspecting their danger : they are really confirmed drunkards, and yet would repudiate as an insult the idea that they are swayed by cravings for alcoholic beverages, and that their daily use is a necessity. Some would fight at the mere insinuation of this fact, and nevertheless they are hopelessly enslaved. The distressing vice has advanced gradually and insidiously, but surely ; stealthily it has overwhelmed its victims, and though they may not yet realize their unfortunate condition, their friends and relatives understand but too well their desperate situation. Medical experience every day teaches the truth of this assertion. Now, there is evidently a great dissimilarity between the effects of alcoholic liquors upon different individuals. In some they do not appear to have any deleterious influence at all, while in others their effects are most obnoxious and brutalizing, often being the source of many deplorable catastrophes. Then, again, there is a condition produced by alcoholic indulgence essentially different from either of these : for instance, some individuals are more or less under its influence the whole year round ; they are not actually drunk, but are always “boosy ;” their

breath is invariably alcoholic and offensive, and this seems to be their ordinary condition; apparently they are not intoxicated, their walk is steady, they can converse, write, and attend to their business: still, they are ever under the pernicious influence; perhaps once in awhile they experience an attack of acute alcoholism, but not often. Then, again, some men will go on a spree lasting several weeks, drinking profoundly and continuously, and then will swear that they have taken their last drink: they fully appreciate the disastrous effects of drunkenness physically and morally, are ashamed of themselves, have done things which as married men or fathers of families have caused them excessive mortification. They endeavor to reform. Their prolonged spree has perhaps thrown them out of employment; they see the evil of their ways, and are perfectly resolved to withstand all future temptation. They do pretty well, it may be, for several months, and then they start on another course of dissipation, possibly a worse one than the preceding. It seems to be a physical necessity, something they cannot resist, no matter how strenuously they endeavor. This is called *oinomania*. When such a man comes to you, you may be led to believe in his resolutions of amendment, as he manifests such a sincere disgust for his conduct. You will take an interest in him, and will expect him to reform; but you will be disappointed, for the next spree will sooner or later occur, to be followed by others, till the unfortunate man dies after some protracted debauch. I must confess that impatient as I formerly became with such people, they now awaken the utmost pity of my heart, for there is so much tenacity, such a fatality, about their disease, it is so difficult to cure it, that they deserve our sincerest sympathies. These men, oftentimes of high social or religious standing, struggle resolutely and persistently, but finally wake up to find their condition hopeless, and sooner or later they succumb to chronic alcoholism, *oinomania*, or some other form of alcoholic disease, and, if they do not die broken-hearted, soon fall a prey to their excesses.

This distressing disease is just as intractable as epilepsy. You recollect the difficulty of management and the infrequency of cure of epileptic affections: this is precisely the rule in alcoholism. This you will better be able to understand when we come to speak about its pathology.

I have already stated that some persons indulge freely in the use of alcoholic liquors without apparently any bad effects. This is owing to their unusual powers of elimination. On account of these powers being very good, some men are enabled to take alcoholic beverages in quantities that are astonishing and truly marvelous. The elimination takes place by the lungs, the kidneys, and the cutaneous perspiration. Therefore you will find that when men drink excessively they have a tendency to frequent micturition. The blood is so surcharged with alcohol that an effort is made to get rid of it through all the excrementitious channels. You will often smell the alcohol on the breath of such people and yet be at a considerable distance. They are so impregnated by it that its odor is readily detected notwithstanding all the methods resorted to whereby to deceive.

The question now arises, Is alcohol entirely eliminated from the body. I am a firm believer in the doctrine that only about one-third of it is eliminated and that the rest is appropriated. For alcohol itself is a food, and, when taken in some acute diseases, is often excellent both as a remedial and as a nutritive measure of restoration. It is a reliable depressor of temperature, it subserves certain nutritive processes, and may thereby save life. Now, this may seem to be in contradiction to what I stated in regard to chronic alcoholism. I then said that the *prolonged* use of alcohol causes an excessive retrograde metamorphosis, by the blood being surcharged with an excess of fatty matters, and the consequent imperfect oxidation of the tissues. This is undoubtedly true in chronic alcoholism; but in acute disease, when high fever exists, when the patient is on fire, so to speak, alcohol is invaluable, and for two reasons: first, it is a hydrocarbon, capable of being taken up as food, and thereby assisting in preserving the vital force till the inflammatory action has passed; secondly, it prevents the excessive tissue-oxidation which occurs during fever, thereby sparing life when the danger resides in the high temperature; and, next to quinine, there is no better depressor of temperature than alcohol. This doctrine I announce to you as true, and as fully confirmed by personal observation. This depression of temperature is always made clearly evident by the only infallible instrument used in medicine,—the thermometer,—notwithstanding the recent observations of Dr. Ringer of a con-

tradictory character. Quinine is positively the best antipyretic, far more satisfactory than digitalis, veratrum viride, tartar emetic, or aconite, when carefully and impartially essayed. I hope that I have put you completely in possession of this practical fact, for you can readily appreciate its importance. It is of importance to you whether as surgeons, obstetricians, or physicians. You will often have inflammatory fevers and pernicious diseases, such as malignant puerperal fever, that will send the mercury in the thermometer up to 104° or 105° Fahr., and here your only hope is in quinine and alcohol.

Now, I have no desire that you should understand me to say that alcohol ought to be administered in every fever. There are rules governing its use just as definite as those that are applied to any other drug, and it is as a drug that alcohol possesses its undoubted virtues. You know very well that an alcoholic stimulant ought not to be poured into the human system like oil into a can or into a lamp, but that certain therapeutic rules must be observed; and we should never give it in such doses that its pernicious effects would be produced. As regards the definite quantity that will produce bad results, this cannot be stated, for what will affect one injuriously will be beneficial to another. Even ladies take daily over one pint of whisky during severe typhoid fever,—a quantity which, in health, would produce coma, and yet the influence of the liquor in that disease will be barely perceptible. This shows the great amount of tolerance for alcohol, which is most particularly evidenced during the continued forms of fevers.

One of the best rules by which to ascertain an excess of alcohol is by the breath. Whenever I give alcoholic beverages during a disease, either as a food, as a stimulant, or as a means to effect a reduction of the animal temperature, I always watch the breath: should this become surcharged by its vapors, it shows that an elimination is going on by the pulmonary mucous membrane, due to an excess taken which is not utilized by the system, and I correspondingly diminish the quantity of the liquor to be administered. Hence this rule is of great importance.

I have said already that about one-third of the alcohol introduced into the body is eliminated: this, of course, remains unchanged, and no matter how it passes out of the body, whether by the urine or otherwise, it is always as alcohol. But the re-

maining two-thirds are appropriated, and when it does pass out at all it is in a metamorphosed condition, and can be no longer detected as alcohol by that most delicate of chemical reagents, the chromic-acid test.

We shall now inquire into the different causes of alcoholism. We have first the *predisposing* causes; and these we may, with Anstie, divide into *occasional* and *constant*. The *occasional* causes are such as are due to the particular occupation of the individual. Persons who lead a sedentary life, like barbers, and shoemakers especially, are more prone to the disease than others. Again, people who are constantly exposed to the inclemencies of the weather, at the same time lacking the necessary self-control in the use of liquor, very often become chronic inebriates. Among the occasional causes might also be classed mental depressions, as we see them sometimes in hysterical women or men, or in men who are not manly enough to meet the annoyances and emergencies of life without drowning their sorrow in the bowl. During my college career, I had a room-mate about fifteen years old, who occasionally missed a lesson; this annoyed him a great deal, and on every such occasion he took a drink of whisky to overcome his vexation. I have never heard the subsequent history of this young man, but I am fully convinced that he has died of chronic alcoholism. Now, it often happens that when a woman has pains, neuralgic, uterine, or dyspeptic, or some menstrual trouble, as dysmenorrhœa, a physician recommends a little whisky; it is a splendid anodyne, and supposed to be good for everything, for the sick and for the healthy, whether people are too cold or too hot, wet or dry; it has become a panacea for all the "ills that flesh is heir to." This reckless prescribing of stimulants without any definite reason, simply for the sake of giving something, is truly deplorable, and the direct cause of many a case of chronic alcoholism. A physician who does this has a terrible moral responsibility to shoulder: many a practitioner has laid the foundation of future inebriation by prescribing liquor in chronic diseases. Some persons have commenced with small doses and gradually increased the quantity, once the pernicious habit is established, until finally their draughts are enormous. Let me give you a golden rule in regard to this, and I hope you will be always guided by it. Never give alcohol in any *chronic* form of

disease, in any continuous pain, in hysteria or hypochondriasis: if you do, you may teach the sweetness of the forbidden cup and the delights of alcoholic narcotism. It is true that some hysterical women will importune you every day in your office, and to get rid of them you may be tempted to give them the first thing that occurs to your mind; but do not prescribe a remedy which you may be powerless to discontinue. When you commence to practice, you will find that there are many lady inebriates,—ladies of excellent families, of refinement and of cultivation,—and you may be called upon to diagnosticate and cure some pretended nervous disease when the symptoms of chronic alcoholism are very apparent,—the nervousness, the insomnia, the tremor, the morning vomiting (not of pregnancy, but of the abuse of liquor). Now, if you diagnosticate chronic alcoholism, do not for a moment think of telling the lady that she drinks too much, for if you do you will undoubtedly be accused of insult; but be adroit, use tact, endeavor to obtain your patient's confidence at the same time that, without effrontery, you let her know you understand her case, until you succeed in convincing her that stimulation is to be entirely eschewed in consequence of its great personal dangers for her. Be prudent, kind, and gentle; otherwise you will be overwhelmed in a storm of fury, and will accomplish no good, but much harm.

Another class of people come to you and complain of vague or indefinite pains. They will say, "Doctor, I do not want you to give me anything to drink, for it may create an appetite I deplore, and produce a habit over which I may have no control." These people appear to be very conscientious, and do not wish to assume any responsibility: they would rather shift it upon your shoulders. After talking a little while, they will ask, "But, doctor, do you not think that a little whisky would be just the thing for that pain?" Never say yes. Never help such people on to the road that leads to ruin. Never assume such a responsibility; be firm, and invariably answer no; and, if necessary, explain your reasons for so doing.

The constant predisposing causes of alcoholism are: *first*, the hereditary transmission of drunkenness in a family; *second*, the transmutation of nervous diseases, of which I have spoken so frequently, and which, as you all know, is a change in form of

different nervous diseases occurring in different generations of one family, so that we find children of epileptic parents to suffer from chorea, epilepsy, alcoholism, insanity, etc. These unfortunate individuals are subject to alcoholism; they bear it in their blood from the moment of their birth; the tendency to the disease exists, and the first taste leads almost inevitably to uncontrollable and excessive indulgence; no matter how skillful, you cannot prevent the direful results. Many authors assert that when the father is in a drunken condition during the fruitful sexual act, the child will necessarily be an incubate sooner or later, or be subject to some form of mental alienation. The struggle to shake off the influence of this hereditary transmission is as sad to witness as it is oftentimes unavailing.

LECTURE XXV.

ALCOHOLISM—*continued.*

Symptoms of Chronic Alcoholism.—Symptoms of Acute Alcoholism.—Diagnosis.—Prognosis.—Pathology.—Treatment.—Beef-juice.—Capsicum.

GENTLEMEN,—If there was one conclusion that we came to in my last lecture, it was that whisky, brandy, or any alcoholic liquor may be given so as to produce different effects, and that these effects will vary according to the quantity of liquor used,—a small quantity proving exhilarating and stimulating, a larger dose being depressing and sedative, an excessive one acting as a narcotic. When we come to study the phenomena of alcohol by the sphygmograph, we shall see its peculiar effects upon the pulse. On the sympathetic nervous system small doses of alcohol produce arterial tension by a healthy irritation of the vaso-motor nerves, while overdoses produce a paralysis of these nerves, which is manifested by the peculiar flushing of the face and the hyperæmic condition of the entire surface of the body. The fact that alcohol in sufficiently large doses produces narcotism was taken advantage of in olden times during surgical operations. This happened before the introduction of anæsthetics in operative surgery, and the operations were rendered more or less painless by inducing profound sleep, or by stupefying the patient by means of liquor. Even at the present day women resort to alcohol as a therapeutic measure in hysteria and neuralgia; they do not confine themselves to stimulating doses, as they should, but take it in sedative, and even in narcotic, doses. These persons seek this influence because they love it, and, as with chloral, opium, or any other hypnotic, they take it for its effects, and it is the *narcotic* effect which they especially desire.

We shall now consider the *symptomatology* of alcoholism. You will remember that I divided the disease into an acute and a chronic variety. The symptoms of *chronic alcoholism* first claim

our attention. The first thing that strikes us is the characteristic disturbance in the motor functions. We notice this in the jactitation, the restlessness, the impossibility of the patient's keeping quiet, the nervousness and incessant muscular agitation; and as the alcoholism becomes graver this muscular tremor increases correspondingly. It also exists in nearly all the forms of acute alcoholism, as in delirium tremens; though we may have the latter condition without the tremor; but in chronic alcoholism it is nearly always present in greater or less degree. Then there is the persistent absence of sleep,—insomnia; and this is common to all forms and types of alcoholism. It is the constant, the unequivocal symptom, and first develops as a result of the condition of restlessness. There are as yet no brain-symptoms, no hallucinations or mental disquietude, but as the agitation of the system advances the want of sleep increases, and then these symptoms make their appearance. They are peculiar and characteristic: the patient becomes peevish and irritable, and his intimates are sufficiently familiar with these phenomena to know that he has been drinking again, perhaps after a certain period of abstinence. And as these mental symptoms develop, we find a certain want of tenacity of purpose, an indecision, an inability to finish anything the individual may undertake, and his mind is never at rest.

We have in the first place the muscular agitation, then the mental perturbations, the irritability, want of decision of purpose, etc. Should the condition still become aggravated, certain vertiginous symptoms will supervene and complicate the situation. The person now becomes dizzy, not from a loss of consciousness, as in epilepsy, but from gastric disturbances; and finally we have the morning vomiting, of which I spoke in my last lecture. It has been adroitly argued by persons who believe in the older doctrines, which I hope I have disproved to you as baseless and foolish, that this vomiting, as well as delirium tremens, is produced by the recoil of the system on account of the withdrawal of the accustomed stimulant. This is all nonsense: the morning sickness occurs because the individual is full of alcohol; he is ill and feels miserable, his normal functions are in abeyance, and the vomiting is merely a nervous symptom of the disease. But, it is said, he does need a stimulant, and he is restored again after taking a dram of whisky. Gentlemen, a cup of strong coffee, or

of beef-tea, will have corresponding effects. The nutritive powers are depressed by the abuse of alcohol, and a stimulant is needed; but there is none better than meat-juice, the most natural stimulant, it being a highly nitrogenized and easily-digested substance. I have not time to dwell any longer upon this morning sickness, but shall merely state that it is a pity that persons thus afflicted do not have an evening sickness also, or are not sick all day long, before they succeed in bringing ruin and disgrace upon those who have the misfortune to be connected with them.

Following this condition is one of visual and auditory hallucination: the patients see and hear horrible things, are frightened and restless. There are no true delusions, as in insanity, but these may come later. I have now such a patient under treatment, and when I visit him and ask him how he slept, he answers that he did not sleep at all, owing to a lot of black cats in his room, that he is afraid of them, that they jump on his bed with glistening eyes, mewling, purring, and making an infernal noise. I asked him once if he believed this, if he positively thought these cats were in his room. He grew quite indignant, and asked me if I considered him a lunatic. Now, here you see the plain difference between a hallucination and a delusion. This patient knew that all he heard and saw was only the product of a morbid fancy: hence he had no delusions and was not insane. But previous to this state we notice loss of sleep, frightful dreams, and certain scintillations of light before the eyes, *muscæ volitantes*, etc. These symptoms are the immediate precursors of hallucinations. Regular toppers cannot sleep, for several reasons. *First*, on account of the constant muscular agitation; *secondly*, because of the hyperæmic state of the brain, especially in advanced alcoholism; *thirdly*, because if they do get to sleep they are scared by horrible dreams. They get up physically dejected, and then commence vomiting. After that they take a drink of whisky as a prelude to the course of narcotism which is the regular plan of the day. And when their intemperance is confirmed, they take their morning drink before getting out of bed, keeping a bottle under the pillow. In some cases which I have treated, a bottle had become insufficient, and the patient would keep a demijohn near the bed. I knew a man in this city who kept a demijohn of whisky within his reach, and when his forces commenced to fail and he began to

grow weak, he placed the demijohn on a chair in a tilted position, so that he might continue to indulge in the degrading vice, which he did till death ensued.

Sometimes symptoms of insanity are developed, for, as I have previously stated, alcoholism sometimes results in insanity; and not long ago a man who is still insane threatened to blow my brains out because I gave a certificate of insanity in his behalf, when, as he asserted, he was only drunk.

We have another set of symptoms to consider, the *gastric symptoms*. Besides the vomiting we find anorexia, dyspepsia, constipation, and pyrosis: these occur in consequence of a hyperæmic condition of the digestive organs, caused by the repeated use of concentrated alcoholic drinks.

As alcoholism advances, the individual becomes more and more degraded: not alone the mental but also the moral faculties become impaired, and you may note it down as a fact that chronic inebriates are generally confirmed liars and arrant cowards. It is in this manner that noble specimens of the human race often become debased by the repulsive vice. Sometimes they become subject to epileptic attacks, or to apoplectic seizures, the sensibility is impaired, and the co-ordination of muscular movements is disturbed, giving them the appearance of people suffering from locomotor ataxia, so that alcoholism might be mistaken for this disease; and not unfrequently it does result in true sclerosis of the cord. Again, there exists in the minds of these patients some vague apprehension of an impending calamity: they come to you saying that they are afraid to walk on the streets, that everybody seems to be pointing the finger of scorn at them, that something terrible is about to happen; and their fright often becomes so intense as to be harrowing to witness. When delusions appear, when they really believe the hallucinations, their condition is much worse, for we then have chronic alcoholism *plus* insanity. I had a case recently in the asylum, in which the patient was very restless, and could not sleep; he imagined that a fellow he called "John" was hiding under the bed, and he said that the reason he did not sleep was that this individual pulled his testicles all night as soon as he was unconscious. This was no hallucination: the man firmly believed it: it was a delusion. He remained in the asylum for a few months, and then was discharged cured.

There is one more characteristic,—the *peculiarity of the appearance*: the countenance is stolid, the look is one of mental hebetude, there is a brutal coldness in the aspect, the features are dull, the face is flabby, and the muscles of expression are no longer normally co-ordinated; the nose is big and turgescient, and acne rosacea appears in the neighborhood of the lips and nose; the conjunctiva is reddened, the eyes are watery, the breath is foul, and the entire face is flushed and congested.

We shall next consider the symptoms of *acute alcoholism*. I have told you that under the head of acute alcoholism we have *delirium tremens*, *oinomania*, *acute mania from alcohol*, and *acute melancholia from alcohol*. Delirium tremens you cannot mistake for anything else: it is self-evident, and is apt to supervene during chronic alcoholism after a prolonged debauch,—not, as I have repeatedly told you, from the mere withdrawal of the accustomed spirituous drinks. Delirium tremens may occur, though rarely, in persons not accustomed to the use of alcohol, as in a young man who has been on his first big spree; but usually it appears when a man has been more or less accustomed to excessive drinking. This condition is usually developed in the following manner. First the patient cannot sleep, and then a “busy delirium” sets in,—not of an asthenic form, as in typhoid fever, but “good-natured and loquacious,”—the patient talks to you about the hallucinations that prevent him from sleeping. The pulse is rapid and feeble, the skin is cold and clammy, yet there is no fever. The visual and auditory hallucinations continue: the patient hears dogs barking at him, and sees rats and snakes running over his bed. In these cases, if not relieved, the tendency is always to the development of a low, adynamic state, and if not cured the individual dies from asthenia. Symptoms of a typhoid character supervene: the pulse is feeble, the vital powers are prostrated, depressed, and exhausted, the tongue is dry and cracked, sordes collect on the teeth, and the patient sinks into coma and dies. Sometimes he suddenly sinks without all these symptoms: hence the prognosis in delirium tremens should always be guarded, for it is dangerous in proportion to the frequency of its occurrence, the depressed condition of the vital powers, and the complication of some intercurrent disease: pneumonia, especially, is to be dreaded. In most cases the muscular tremor exists, and when it disappears it is a

sign of improvement ; but sometimes it does not occur at all, and delirium may exist without tremor. Do not forget that after exhaustion from alcoholic excesses, with long and protracted insomnia, there is a tendency to the development of typhoid symptoms, and that the patient's condition then becomes critical.

Oinomania and some other forms of acute alcoholism I shall not dwell upon, having described them in my last lecture.

Acute mania from alcohol is apt to be developed in persons who have a predisposition to insanity by hereditary transmission and indulgence in intemperate use of alcoholic stimulants. It is easily recognized by the development of delusions and homicidal propensities such as occur in ordinary mania.

Acute alcoholic melancholia is known by great depression of spirits, etc., and by other manifestations of insanity with which you are all familiar.

How are we to diagnosticate alcoholism? I have given you the more important symptoms in connection with the different forms of the disease, and I have now simply to state that by grouping them in any given case you will experience little difficulty in avoiding fallacious conclusions. First of all, as in every other disease, ascertain, if possible, the history of the case. You cannot always, however, obtain this record of a person's habits. You will recollect that I spoke to you of females, who sometimes complain of some vague nervous affections, seeking your advice, but at the same time trying to keep you in ignorance of the true cause of their illness. They will not admit that they are addicted to the use of liquor : they carefully conceal their bad habits. Here it is your duty to suspect and discover the true cause. The symptoms of chronic alcoholism already given, such as muscular tremor, morning sickness, flabbiness and a congested appearance of the face, undue development of adipose tissue, the peculiar want of harmony in the action of the muscles of expression, the development of acne on the face, the insomnia, the mental disquietude, the vague apprehension of the advent of some calamity, the greater or less deterioration of the moral faculties, constitute a picture not to be misinterpreted. The visual hallucinations concerning snakes, dogs, and cats, and such other morbid fancies, are peculiarly distinctive and characteristic of alcoholism.

The *prognosis* of alcoholism is most unfavorable ; that of de-

lirium tremens, when judiciously treated, is very hopeful. When religious motives and moral efforts fail, it is certainly difficult for a physician to effect a cure. If a physician succeed in gaining a patient's confidence and in making him follow the directions, the patient may be saved; but, unhappily, this is not always practicable, and a good many will not submit to the total-abstinence plan. Then some will take a drink at the slightest annoyance; others will stop for a month, or a year, or even several years, apparently cured, and at last one single glass of wine may shipwreck them for life. I knew a man who had not tasted liquor for many years, but who, meeting a friend whom he had not seen for a long time, was persuaded to drink only one glass of sherry: that single glass was fatal in its effects, and once more awakened in him the ravenous desire for liquor; the morbid taste was more intense than it ever had been before: he drank more recklessly than formerly; and finally he died a miserable death, after dragging his family from easy circumstances to the depths of poverty.

As a matter of course, if some complication arise, such as a disease of the liver or of the brain, the case becomes more hopeless. However, these complications do not occur so frequently as is generally supposed. When you look into your books, you will find it stated that cirrhosis of the liver is generally produced by the abuse of alcoholic stimulants; but I can tell you that, with an enormous experience, I do not recollect more than three or four cases having this termination.

The *pathology* of alcoholism I have frequently alluded to during these lectures, and I shall now recapitulate its principal features. First, the irritative effects of alcohol are produced on the gastric mucous membrane, resulting in an impairment of the digestive functions; there is a decided congestion of this membrane, and a hemorrhage, due to an intense stasis of blood, not infrequently occurs. We all know that alcohol coagulates albumen: hence the idea that digestion is facilitated by the use of alcohol is, I fear, a mistaken one; at any rate, large doses of alcohol deteriorate the gastric juice, producing pyrosis and other dyspeptic symptoms.

I have frequently dwelt on the devitalizing effects of alcohol on the blood,—on its tendency to produce an accumulation of

fatty matter in that fluid, and at the same time divert it from its proper uses. I have told you how the hydrocarbon assimilates the free oxygen of the blood, leaving it in a hypercarbonized condition, no longer able to perform properly the functions of nutrition. The blood being thus deoxidized and surcharged with alcohol, all the organs supplied by it become necessarily impregnated with the poison, and nutrition is greatly impaired.

The third effect is the remarkable selective affinity exhibited by the nervous centres for alcohol: they seem to attract it to themselves whenever it is in the circulation. This is not more remarkable than the affinity of the spinal centres for strychnia, which, given in overdoses, increases the polarity of the cord, producing disastrous results. The nervous centres thus impregnated with alcohol are no longer in a condition to perform their normal functions; and central troubles, such as softening, sclerosis, hemorrhage, epileptic attacks, etc., often manifest themselves.

How are we to treat this disease? If you have carefully followed me in these two lectures, you will surely anticipate a few of my conclusions. First, if called upon to treat alcoholism, you have two classes of cases seeking relief, the acute and chronic forms of the disease. As future physicians, this subject of treatment is to you necessarily a very interesting one. The fundamental fact of which I spoke in my last lecture is the immediate *total-abstinence* plan, which is the *sine qua non* of success in all cases, acute or chronic. This is the only reliable method, and it has proven very satisfactory. In the New York Inebriate Asylum, at Binghamton, a large percentage of cases have been restored. Of course, in asylums where patients come voluntarily and can leave at will, they do not always want to submit to this method of treatment; but if you succeed in gaining their confidence, if you can have your instructions faithfully carried out, you can generally manage to effect a cure. The first thing to do is to discontinue all alcoholic supplies: this is absolutely necessary. Of course, if a typhoid condition supervenes, you should administer alcohol, especially if the vital powers are sinking. It is always easy to give it, after it has been withheld, when its stimulating effect is called for, but it is not easy to do away with its ill effects when it has been injudiciously used. But, you may ask me, suppose I withhold the whisky, and the patient sinks?

Well, gentlemen, if you are afraid to assume responsibility, you must not practice medicine. It is but a matter of common sense, that when the system is surcharged with a poison, no more should be given; therefore total abstinence is the first law you have to follow. But, again, you may say, If I were reduced to this unhappy condition, I would acquire self-control. I would act like a man, and indulge in only three or four drinks daily: cannot the same rule guide others in their action? In reply, I will simply state that thousands of unfortunate individuals daily make the same resolutions, swearing that they will take but a limited number of drinks, and generally before night they are drunk. Remember that in these cases the volitional powers are often at fault. This is invariably so when the love for alcoholic drinks is the result of disease and springs from an hereditary predisposition. Would you expect any insane person to control the desires growing out of his insanity? Total abstinence is the golden rule in alcoholism, as in the opium habit. A most highly nitrogenized and liberally-administered diet constitutes the rest of the treatment, and is absolutely essential. You will be astonished at the wonderful effects of beef-juice, how rapidly old toppers recover under its administration. Besides this, you may add a stomachic stimulant, like capsicum; but, if too great a gastric irritability exists, this is first to be overcome by a few grains of calomel, followed, after the bowels have been freely moved, by subnitrate of bismuth, which is excellent in such cases. In conjunction with these, you may give iced Vichy-water, and even a little iced champagne, on account of the carbonic acid which it contains; and also apply a mustard-plaster to the epigastrium. But as soon as the gastric irritation is subdued, give beef-tea and capsicum. Now, I do not know whether capsicum is advantageous on account of a quieting effect which it is claimed to exert on the ramifications of the gastric nerves, but I am sure that it acts beneficially in many cases.

From experience I am fully convinced that men addicted to intemperance cannot with any safety touch liquor. Their only hope is in total abstinence for life. Even beer and wine will excite an irresistible desire for stronger drinks. An indulgence in a few drops of wine will not rarely do away with the good results of years of perfect sobriety. Moral considerations, the ties of relationship, self-respect, high social position, efforts at

self-control, will all be unavailing and ineffectual unless the law of total abstinence is followed. The best of resolutions will otherwise snap like threads before the hurricane. Always resort to tonics early. Remember the pathological condition of alcoholic narcotism, and you will see that something is required to cause a healthy innervation of the vaso-motors. For this purpose nothing is more valuable than the administration of the bromides and of quinine,—the latter in small doses, say about one or two grains three times a day: its effects in relieving the existing depression are often wonderful.

As regards the insomnia, the first thing I must tell you is, avoid opium, for reasons which I shall soon explain. We have a precious remedy for this purpose,—*hydrate of chloral*; should this fail, give *bromide of potassium*, or combine either with tincture of *hyoscyamus*.

The moral influence to be exerted is of great importance. During the two or three days of intense craving for stimulants, there will be many moments of sadness and dejection, and you must not abandon your patient, but try to keep up his courage by gentle words and kind attentions. If once you gain his confidence, if he feels that you understand his case, he will always cling to you, and even follow you from city to city. But also be firm; never compromise with him; do not permit the use of alcoholic liquors in the smallest quantity; for if you are timid and wavering he will notice it instantly, and reason with you and persuade you against your own convictions. Should you yield, your want of firmness will surely cause him a lifetime of misery, and will forfeit for you his esteem and respect, the patient being himself in his heart convinced that the course of treatment you have been persuaded to abandon is correct. The truly humane method of treatment is to shorten the pain, and this you do by shutting off the use of alcohol at once: the pain will be intense, it is true, but it will not last long, and the struggle, though fierce, will soon be over; while by the old method of allowing moderate quantities of stimulants you will never accomplish anything, and will torture the patient to no purpose. At the same time, you must be constantly on your guard not to be deceived, for the slaves of alcohol, especially in the advanced stages, are audacious liars and very deceitful: they sometimes manage to obtain liquor, and will care-

fully conceal it, and as soon as you are out of sight will commence drinking.

To show you that my conclusions are correct, I shall adduce just one illustration. If any of you were ever great smokers, and have tried to get rid of the tenacious habit, you know that it is much easier to leave off smoking all at once than to try to limit yourself to only two or three cigars a day, in the hope that your desire for tobacco will gradually grow less. This limitation method is a fraud; no man can adhere to it unless he has an iron will, and we do not find many such instances. If you continue smoking, and try to smoke less, the very smell or thought of a cigar will be irresistible. So it is with alcohol,—the taste of it always starts the desire for more; and even when they take it in sedative doses, drunkards always crave more: the very indulgence seems to incite a new desire. I often meet persons in the asylum who remain till they are well and rational, and then go out for another spree: by-and-by they come again, staying till they are once more convalescent; and so they keep coming and going, till they die from a succession of debauches. When they come in, and are not too debilitated, they get a moderate mercurial purge, about eight or ten grains of calomel: if the stomach is irritated I give them soda powders, put a mustard-plaster on the pit of the stomach, and let them eat ice freely. If they insist upon stimulants, I prescribe a mixture of one ounce of tincture of capsicum, two ounces of compound spirit of ether, and two ounces of tincture of valerian (ammoniated), and of this they can take a dose in water frequently repeated. After the stomach is settled I give capsicum in beef-tea, and if the stomach does not retain it I inject the beef-tea, alone, into the rectum. If there be much muscular tremor, I give strychnia in small doses, about one-sixtieth of a grain three times a day; then, as a remedy to act on the vaso-motor nerves, the remedy *par excellence*, bromide of potassium, which controls existing hyperæmia, with chloral at night to produce sleep. But give everything very cautiously, especially the chloral, as the patients sometimes die very suddenly. Do not give more than about fifteen grains of the latter every two hours at night, and, if the insomnia is obstinate, combine the chloral with bromide of potassium and with hyoseyamus; but never give opium.

LECTURE XXVI.

ALCOHOLISM—*concluded*.—HYSTERIA.

Treatment of Acute Alcoholism.—Opium.—Rich Hot Broths saturated with Capsicum.—Bromide of Potassium.—Ether.—Chloral Hydrate.—Complications.—Hysteria.—Hysterical Epilepsy.—Divisions of Hysteria.—Psychical Hysteria.—Motor Hysteria.—Sensory Form.—Hysterical Hyperæsthesia.—Hysterical Anæsthesia.—Pathology.—Treatment.

GENTLEMEN,—The last time we met, I promised that to-night I would take up the subject of the treatment of acute alcoholism. A good many physicians are indoctrinated with the old view that delirium tremens (as it was formerly described) is an affection aggravated and prolonged by a want of sleep, which, therefore, it was necessary to produce *at all hazards*. Now, I shall commence by attacking this proposition, and state, first, that although delirium tremens is sometimes relieved by sleep, it is nevertheless an undisputed fact that some patients do not recover even after a continued and prolonged sleep has been induced. On the other hand, delirium tremens is not a disease in which sleep must be produced at all risks and by all means, for the reason that it is strictly self-limiting in character, like chorea and some other diseases. The insomnia is but a symptomatic indication, and by no means constitutes the disease proper, which is peculiarly prone to run its course, not necessarily being influenced by remedies: still, the duration is not definite in every case, and may be subjected to changes on account of different influences. At all events, it is a positive fact that there is a certain duration of the disease before the patient reaches that state of health which he enjoyed previous to the attack. Now, in regard to the treatment, we have been told that opium is our sheet-anchor, the only means by which to achieve our purpose,—that we must give it *coup sur coup*; and, as Dr. Wood in his work recommends, two grains of powdered opium every two hours till sleep supervenes. Now, gentlemen, if you should pursue such a course it would be in

direct opposition to the recommendations of some of the best lights of modern science. When we study the effects of opium, we ascertain that it exerts a most pernicious effect upon the ganglia of the great sympathetic nervous system. Sometimes, even without producing a loss of consciousness, we may by repeated doses of the anodyne so depress the organic nerve-centres as to produce disastrous results; and according to the kind of nerve-centre most affected will we have a narcotized condition of the vital organs over which they preside, and the patient may perish from the deleterious effects of opium upon the ganglionic nervous system. Then we have its deleterious action upon the digestive functions, and we know that these have already been reduced to a sufficiently low vitality by the long-continued and excessive use of alcoholic liquors; and we are aware that the mucous membrane of the stomach and that of the duodenum are already in a state of intense congestion, with corresponding gastric disturbances. But if we give opium we only add to the difficulties, and, as we have already sufficient anorexia, we only increase the trouble, so that we augment the already existing vomiting by the opium, in this way greatly diminishing the most valuable means we possess to save the patient's life, which is the retention by the stomach of healthful and nutritious materials. You undoubtedly remember that in my last lecture I spoke of good nutrition as the remedy *par excellence* in these cases. But, fearing that you may imagine that I overstate the facts, or that it is owing to some deep-seated prejudices that I praised this particular method, I shall quote you the words which Anstie (the celebrated author who wrote on stimulants and narcotics) uses in this regard. He says, "The typical member of stimulants is simply easy-digested food, and the successful treatment of delirium tremens, in nine cases out of ten, depends on the regular and continuous supply of suitable nourishment, whereby the functions of the nervous system are supported during the struggle towards recovery." Now, independently of this fact, opium, by its depressing influence on the centres of organic life, sometimes has a pernicious effect by inducing a paralytic condition of the ventricles of the heart, and the result is death from cardiac syncope; the patient becomes deadly pale, the radial pulse fails, the temperature sinks, the circulation is suspended, and death ensues.

Hence it is not a specific in delirium tremens, as has been asserted, but should be only exceptionally employed. Now, this may seem to be a bold statement, and even in contradiction, perhaps, to the results obtained by some of you in your own experience; but it is true, and, as I said, in nine cases out of ten a regular and systematic nourishment will generally relieve the symptoms. Recollect that as in chronic alcoholism, so in this case, food of a highly nitrogenous character, such as rich broths, given hot and saturated with capsicum, is invaluable. Again, when treating delirium tremens, provided there be no development of asthenic symptoms, or of a typhoid condition, bromide of potassium will be found to be an excellent remedy, and very efficient in allaying nervous irritation. Ether may also be recommended in this connection. You can also try to procure sleep for your patient; but always do so with caution; and of all the remedies which may be used for this purpose there is none equal to hydrate of chloral. Now, in reference to the old treatment by tartar emetic, digitalis, chloroform inhalation, etc., I shall merely say that I deprecate these remedies, and the discussion of their merits I would regard as a loss of time. But I wish that you would recollect this one word of caution: when you administer hydrate of chloral you should never give it in injudicious doses, as you may kill your patient. Always remember that delirium tremens is a self-limiting affection, and that if you persist in your efforts at producing sleep it is not without imminent risk of sacrificing the life of the individual, promoting a deep and lasting sleep, out of which he will never awake. In order to prove that narcotism does not constitute *per se* the treatment of delirium tremens, I shall again quote Anstie. He says, "The idea that patients in delirium tremens require to be narcotized into a state of repose may now be said to be abandoned by those *best qualified* to speak." Nothing can possibly be more precise.

As to the question of the propriety of stimulation, recollect, in the first place, that I told you that in delirium tremens alcoholic stimulants are simply out of the question. From moral and from medical points of view I entirely disregard them. Moreover, by the administration of alcoholic stimulants you encourage the use of the very substance the abuse of which has brought about the sad condition, and even in old inebriates it is the last thing of which I

think. But, as I told you when speaking of the treatment of chronic alcoholism, so in delirium tremens, should the withdrawal of liquor induce a typhoid condition, then, of course, you may stimulate with alcohol; not otherwise. As this is a most important subject, and diametrically opposite to the treatment adopted by many physicians, I once more quote Anstie: "In every case, however, I think it is our duty to abstain as long as possible from the use of alcohol, and before resorting to a treatment of such doubtful propriety we ought to try less harmful narcotic stimulants." As I have just stated, even in old cases of alcoholism I refrain from resorting to it, and withhold it as long as possible.

Whilst giving good nourishment to the patient, you should see to its systematic administration. It should be given in bad cases every two hours, night and day, with chloral at night to promote sleep. The person should be placed in the best possible hygienic conditions, and, when convalescent, tonics and nervines may be given.

These are the measures resorted to in the treatment of acute alcoholism. And remember this rule: all active and injudicious interference should be avoided. It is not necessary to stop *this* or *that* symptom: the disease is self-limiting, and if you interfere imprudently you not only do no good, but on the contrary positive harm. Many persons with delirium tremens have died from the injudicious use of opium. Should you, however, persist in employing this drug, never give it in any other way than hypodermically, commencing with doses of not more than one-sixth of a grain, and then watch with care and precision the results.

In conclusion, I will remark that acute alcoholism may be aggravated by the supervention of *complications*, such as pneumonitis. Where this occurs, the symptoms are not always active or evident, but mostly latent, insidious, masked; there may be but little cough, and hardly any expectoration, and yet the pneumonia may be very grave. This complication being of rather common occurrence, it becomes necessary to auscultate the chest in every case, lest death should occur through the pneumonia not being recognized by the physician. In chronic alcoholism, galloping consumption is sometimes developed, carrying the patient off in an extraordinarily short time. I recollect the case of an inebriate whom I had charge of for many years, who once sent for me during

an acute exacerbation of alcoholism. Previous to this last attack his health was very good, and he was always able to pursue the ordinary avocations of his life, which consisted mainly in open-air exercise. I went to see him, and found that he had been confined to bed for only ten days, and had not previously summoned me because, as he said, he was ashamed of himself. He had been coughing slightly for about six weeks, and upon auscultation I found a large cavity in the apex of one of his lungs, with very few rational symptoms of phthisis. He died about one week after this visit. Thermometry is of invaluable aid in the detection of such complications.

HYSTERIA.

The next subject I shall speak of is *hysteria*; and I shall be brief, as this disease must be studied by personal observation, being peculiarly difficult to describe. When I take up its consideration I always feel discouraged, as volumes might be written without giving adequate ideas of its character, its peculiarities, and its tendency to assume different forms. It is an affection of protean type, and leads many good diagnosticians into most mortifying errors. Hysteria is really a *disease of the nervous system*, and consequently not an imaginary ailment. It has a tendency to certain paroxysmal exacerbations, which are often convulsive in character. The first thing, which is present in nearly all cases, and to which I fully alluded while speaking of certain forms of palsy, is the hysterical constitution. You all remember some of the characteristics of the latter, such as the *clonus hystericus*, the *globus hystericus*, the passage of large quantities of limpid urine, the preternaturally mobile state of the emotions, the facility with which the patients laugh and cry, and the great agitation into which the nervous system is sometimes thrown by the slightest source of irritation. Again, hysteria is an affection which is very prone to assume certain hyperæsthetic or anæsthetic characters, and when we are treating these cases it is often the manifestations of the hysterical constitution which enable us to suspect their true nature, and were it not for certain of its well-known features we should be at times scarcely able to reach any satisfactory solution of the symptoms. If these conditions of hyperæsthesia or anæsthesia develop themselves, they deserve our careful attention.

Sometimes the organs of special sense may be affected and become most acutely sensitive. Some hysterical women will appreciate minute differences in weight and other physical properties of different substances merely by the tactile powers, as if endowed with supernatural faculties. Again, in the hyperæsthetic form of hysteria you will notice a remarkable and *morbid craving for sympathy*, and the patient feels quite happy if the whole family be upset by witnessing the outburst of some simulated or greatly exaggerated nervous manifestation. It is on account of this craving for sympathy that they have such inveterate hysterical coughs, dyspnœa, sneezing, vomiting, etc. Sometimes the disease will simulate peritonitis, or some other grave inflammatory affection of the internal organs, of the abdomen, or chest, or pelvis, and you might be led into error, did you not recollect that there is no inflammation without an increase in temperature: this is invariably absent in all forms of hysterical neuroses, and hence the thermometer will in most instances settle the question, taken in connection with the great superficiality of the localized tenderness and the general symptoms. Sometimes hysterical persons will simulate some serious disease of the joints, and even eminent surgeons, like Sir Astley Cooper, have been in doubt as to whether there was an organic affection of the joint or merely an hysterical manifestation. I know a young girl who was treated for hip-joint disease, in this city, by prominent physicians; she kept her bed for a long time, and all at once jumped up cured suddenly. Of course she had had no hip-joint disease, but simply hysteria; the physicians had mistaken the hyperæsthetic condition of the joint, the agonizing pain upon pressure, etc., for the former disease, and she had been kept in a state of perfect immobility for months, with little or no result. Just as we found was the case in hysterical aphonia, so is it the nature of all hysterical affections to disappear as suddenly as they appeared. This is truly remarkable, and, as I told you of hysterical palsy, it may happen that a woman of a hysterical temperament will go to bed in perfect voice, and the next morning will not be able to utter a sound. So it is with joint-diseases: a woman may be instantaneously seized with symptoms that closely simulate those of organic disease, and a physician may be easily misled, especially if he relies too implicitly upon the statements of the patient. But if the house should catch

fire, or the patient fall in love, or some emotional affair greatly influence her, a sudden cure will be effected, and the disease will end as rapidly as it commenced. The word *hysteria* is taken from the Greek *ὑστέρα*, which signifies a “womb;” but this name is unfortunate, for if a person when thinking of hysteria always associate it with some abnormal condition of the uterus, he will necessarily have a wrong conception of the malady. Many women are hysterical and still do not suffer from any uterine diseases; and, again, some men are prone to hysteria and suffer from many of its manifestations. You thus see why the name is unfortunate. In women, hysteria does not necessarily presuppose even any menstrual derangement, such as dysmenorrhœa, amenorrhœa, etc., or any uterine trouble whatever; but if there be a peculiar susceptibility to nervous diseases, then any other illness or abnormal condition of the genito-urinary organs is very apt to produce hysteria, or to aggravate it should it already exist; but, for all that, many women are hysterical with perfectly healthy pelvic organs. Some authors, however, and, I believe, Niemeyer among them, claim that hysteria is very often the result of some uterine disorder or some anomaly in the menstrual functions.

Hysteria generally occurs during the period embraced between the advent of puberty and the change of life. We have this climacteric period in the male as well as in the female. I told you this when speaking of insanity, and that this change of life in man is often preceded by some very libidinous tendencies; as impotency approaches there will often be exacerbations of the sexual desires. Hysteria is most apt to develop itself at the time of the commencement of puberty in both sexes, but is more particularly a disease of females: it generally ends about the time of the menopause. But it is true that there are many exceptions to this rule; and I remember a child only about eight years of age in whom the hysterical condition was well developed and very decided, and still she was not near the time of puberty, there being no evidence whatever of any attempt of nature to produce menstruation; the mammæ had not assumed the least prominence, and no growth of hair had as yet commenced to cover the pubes. Sometimes hysteria complicates other affections of the nervous system, as epilepsy or insanity, and we therefore have certain complicated varieties of hysteria.

That at puberty the efforts of nature to produce the necessary maturing changes in the human organism are greater in the female than in the male, and consequently attended by greater nervous disturbances, there can be no doubt. I do not concur with some authors, who claim that it is the influence of the different training which girls receive that makes them more liable to hysteria than men. I believe that it is an incontestable fact that the proneness to the disease is always more inherent in women than in men.

Hysterical epilepsy, often associated with pelvic troubles, is curable if we can remedy the primary affection. But if the hysterical epilepsy is once permanently established, it is very apt to afflict the patient for life. These cases yield best to bromide of potassium with iron, and to such measures as tend to the improvement of the general health.

The division of hysteria which I shall adopt is that given by Niemeyer. You will recollect that we divided epilepsy into three forms. So we do hysteria. These forms of hysteria do not exactly correspond with those of epilepsy, but there is a certain analogy which deserves notice. Epilepsy, you remember, we divided into the convulsive, vertiginous, and psychical or mental forms. Hysteria we divide into the motor, sensory, and psychical varieties. Hysteria seems also to have some particular effect upon the circulatory apparatus, and on the nervous system of organic life; but you can take all the varying and protean symptoms of the disease, and they can be grouped under one of the three divisions. If I were to describe all the possible symptoms and manifestations of hysteria, I should greatly fatigue you without exhausting the subject, for no other disease is so prone to assume so many different varieties, and one day you may have one type, the next day another, etc., all in the same patient. *Psychical hysteria* is evidenced by a disturbance of the emotional faculties. In *motor hysteria* there is always a tendency to the production of convulsions, which generally are clonic in character, though tonic convulsions exceptionally occur.

In view of the prognosis and treatment, it becomes important to recognize hysterical convulsions. Now, suppose you were called to see a lady thus affected, the first question which would suggest itself to your mind is, Is this epilepsy or hysteria?

The patient may have clonic spasms, tear her hair, beat her breast (not hard enough to hurt herself, however), and laugh and weep alternately. You say it is hysteria. So it is, of course, and everybody knew it long before you entered the room. But, on the other hand, suppose a woman tumbles: it is generally on a soft spot,—not in the fire,—hysterical women do not fall where they can hurt themselves; epileptic patients fall anywhere when seized, no matter how great the danger that threatens them, even were the abyss of hell to open yawning at their feet, but hysterical women would be more apt to fall into the arms of some gentleman bystander. But to come back to our illustration: the woman falls; a bloody foam may possibly escape from her lips; she has purposely lacerated her tongue; she has opisthotonos, or pleurothotonos, or emprosthotonos. The whole house is topsy-turvy, and if the woman be married, and her husband has never witnessed any analogous scene, he is half distracted,—thinks his wife in a most deplorable state, or that she is actually dying. Everybody is full of anxiety, sympathy, and regret,—which is exactly what she revels in, and if she could keep up this display she would continue to frighten the bystanders for an indefinite period. Now the mother or the husband rushes for the physician and propounds the plain question, “Doctor, is this epilepsy?” Before you answer, recollect the univocal, pathognomonic, characteristic symptom of epilepsy,—the abolition of consciousness. This we find ever present in epilepsy, whether convulsive, vertiginous, or psychical; but such is not the case in hysteria. There may be a partial suspension of consciousness, it is true, in some bad cases of the latter, for all its manifestations are not assumed, though a great deal *exaggerated*. The pathology of the disease teaches us little or nothing, and consequently we do not know what part of the nervous system is at fault; but the entire display is not simulated, that is certain.

Now you know how to recognize hysterical paroxysms; but how are you going to treat them? The case may occur in an aristocratic family, and you may want to avoid offending the lady, in which case it is difficult to advise you; but if you desire an efficacious means of restoration, cold water dashed in the face from a distance is certainly not to be neglected. I have generally found the severity of the symptoms to diminish *pari passu* with

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the amount of water used, the force with which it is projected, and the firmness of the attending physician. For this little exhibition may be kept up for half the night for your especial benefit, unless you are anxious to terminate the scene without delay. If you wish to intensify the phenomena, all you have to do is to exhibit sympathy and tenderness, and you will find a corresponding increase in the violence of the storm, and with every such indulgence there will be a renewed outburst. No, gentlemen, I repeat that there is nothing like the cold-water treatment, used *secundum artem* and *pro re nata*.

I have described the peculiarities of the mental and motor forms, and now I shall consider the sensory variety. This we subdivide into the *hyperæsthetic* and the *anæsthetic*. But before describing them I shall make one remark. Niemeyer relates a case that came under his own observation which will illustrate the marked craving for sympathy existing in hysteria. The patient was a hysterical woman, who had purposely injured a part of her limb so badly as to necessitate an amputation. After the operation had been performed, and the stump had healed, she commenced again to irritate it in a serious manner, evidently to increase the sympathy towards her for which she had such a morbid longing. But let us consider the hyperæsthesia. This may affect any part of the body,—any region, muscle, joint, or extremity; and it may be so intense that the slightest pressure upon the affected part will cause the most excruciating pain. I have already told you that hysteria sometimes simulates peritonitis, and the pain in the abdominal region will be so unbearable that the patient will not allow you to use manipulation. But we have here a remarkable clinical fact,—namely, that in peritonitis *hard* pressure upon the abdomen is simply out of the question, while in hysteria, though *slight* pressure is very painful, deep pressure can be borne, and this is characteristic of the latter. Here you notice what I have repeatedly alluded to before,—the *superficiality* of the hysterical symptoms. Again, the hyperæsthesia selects certain localities,—has peculiar lurking-places: thus, we often find the pain under the left breast, or in the cranial region,—the *clavus hystericus*; or the pain may be in the back, and simulate organic disease of the spine; or it may be in the right iliac region; or we have neuralgic pains, in many cases, in a great variety of situations. The neuralgia is generally

of the fifth pair and of the cervico-occipital nerves, and the cause probably resides in the anæmic condition which more or less complicates and aggravates all cases of hysteria.

When anæsthesia is developed, you may easily be led into error, and imagine that the patient is suffering from some terrible form of paralysis, as in this condition there may actually be a complete loss of sensation in some particular part, and the pricking of pins or needles will produce no pain. This condition may extend to the mucous membranes, and then the individual will swallow lead-pencils, and all sorts of crude materials, without any distaste or gastric disorder. This shows how profound this hysterical state of anæsthesia may be; and Niemeyer, who gives us so many striking illustrations, tells of a hysterical patient who was scared by a hot iron all along the back and did not even wince! This surely proves that hysterical affections are not entirely imaginary, and that in some cases a true cutaneous anæsthesia exists. But, in treating a disease of any organ in a hysterical woman, do not allow yourself to be misled by her deceptions. "Forewarned is forearmed." To show you how deceptive some cases are, I will give you an illustration. Some time ago, in this city, there was a woman who created quite a sensation in the medical world, supposed to suffer from extra-uterine foetation, and her condition was so extraordinary that many physicians visited her. She passed foetal bones both *per vaginam* and *per rectum*, and was examined by many, who were all convinced of the truth of the phenomenon. But among the learned gentlemen called in consultation there was an illustrious surgeon, Dr. Hodgen, professor in the St. Louis Medical College, who was rather skeptical. At his first visit he examined some of the supposed foetal bones, and immediately pronounced them birds' bones, and, after returning home, washed off the fecal matters and found them to be chicken-bones, which she had introduced into her vagina and rectum, thus deceiving many who were more credulous and less cautious. The same patient was some years later admitted into the St. Louis City Hospital with a large cicatrix upon her abdomen, showing that some doctor had been duped into making an exploratory incision.

It has often been stated by different authors that hysteria is generally developed in spinsters, widows, or women who live separated from their husbands, and that it is the unrelieved crethism

of the sexual organs that produces the hysterical manifestations. This view has caused many older writers to advise marriage as a means of curing hysteria in single women. But I am far from concurring in this opinion, considering it an injustice to many pure and noble women to misjudge them in this respect simply because they are hysterical. Hysteria is an affection not uncommon in prostitutes, and nobody doubts that their erotic faculties are sufficiently developed and satisfied. Again, many husbands will tell you that, far from being sexually very excitable, their hysterical wives are disagreeably cold and indifferent. We therefore can be sure that passion has nothing whatever to do with this disease.

I shall be brief in regard to the celebrated *bed cases of hysteria*. You may have heard of persons, generally women, who were confined to bed during a period of many consecutive years,—twelve, fourteen, or more,—supposed to be suffering from some incurable disease. They ate and slept well, their nutritive functions were splendidly performed, and no part of their body showed any sign of atrophy, the disease being generally referred to spinal origin. I have once before had occasion to tell you that when some physicians are treating a brain-trouble which they are unable to understand they always call it congestion; and so it often happens that when a person suffers from some vague nervous phenomena rendering him or her—usually *her*—apparently helpless and bedridden for a long time, a spinal trouble is diagnosticated. Then, after some charlatan or mesmerizer has been called in and made a few mysterious passes, the patient is cured, and the doctors are put to the blush. Of course he has not relieved any organic affection, because it never existed, but has simply relieved hysteria by the mental impression made upon the patient. Some of these people want to become well, and some do not. I heard of a case once of hysterical paraplegia (not the case to which I alluded the other night). The patient had been bedridden for a long time, with no tendency to recovery. It happened that she had a younger sister, who was to be married. The ceremony was to be a grand affair; every one was on the *qui vive*, and preparations were being made on all sides. It was noticed that about a week before the appointed time the patient appeared rather gay and spry; so her physician anticipated a speedy cure. He was careful, however, not to apprise the mother of his suspicions, lest her daughter

might find it out and change her mind. She had ascertained that her old beau was to be there; and on the day of the wedding she suddenly recovered, danced with him all night, and was entirely restored. We find many such cases, where the patients would remain in bed until their death if something extraordinary and unexpected did not happen to stimulate their volitional powers and make a powerful mental impression, thereby producing a remarkable change and restoration.

Should you go into the country to practice, some old women will come to you and say, "Doctor, here is Mrs. A., who was sick so long, and employed Drs. S. and J. and B., men who pretend to know something, but they did her no good, although they treated her faithfully and energetically; she only got worse. Then she obtained the services of Dr. T., who, they say, is not a regular physician, having been a barber or a carriage-maker all his life; but, at all events, she at once recovered, and has been perfectly well ever since." Of course she had hysteria, and the fortuitous circumstance of wishing to get well, combined with the novel influence of the charlatan upon an impressible nature (such as hysterical people always have), eventuated in a disappearance of the disease.

The next point is the pathology. It is something about which we know nothing, and it may be that in hysteria, as in chorea, there is a perversion of the nervous functions from some profound nutritive errors; but it is useless to enter upon a field of hypothetical disquisitions.

The treatment requires that you should observe the same rule that holds good in the treatment of any nervous disease. Place the patient in the best hygienic conditions. Recollect that there is usually an anæmic state in these cases of hysteria, and, therefore, administer good, nutritious diet, with iron, quinine, or arsenic. I have considerable faith also in the judicious use of the mild forms of electricity. The most important measure, however, is the exercise of a certain *moral control* by the physician, which is as necessary in many other diseases as in hysteria. The reason why inferior practitioners sometimes succeed where bright, scientific, and qualified men fail, is on account of the exercise of this moral influence over their patients, it being a great auxiliary in the treatment of all forms of nervous disease.

One more and last consideration. For the treatment of hysteria during the paroxysm the number of specific remedies recommended is innumerable ; but there are only two therapeutic agents that I will advise,—assafetida and valerian. As Niemeyer says, these remedies do not exert any specific effects in the cure of the disease or of the hysterical condition, but during hysterical convulsions their effects are often remarkable.

LECTURE XXVII.

SPINAL MENINGITIS.—MYELITIS.—SPINAL HEMORRHAGE.—CONCUSSION OF THE CORD.—SPINAL TUMORS.—SPINAL IRRITATION.

Spinal Meningitis: Causation; Symptoms; Pathological Anatomy; Prognosis; Treatment.—Grouping of Remedies.—Myelitis: Symptoms; Pathology; Causation; Treatment.—Spinal Hemorrhage: Causation; Symptoms.—Recapitulation.—Prognosis.—Treatment.—Differential Diagnosis.—Congestion of the Cord.—Concussion of the Cord.—Spinal Tumors.

GENTLEMEN,—To-night I shall speak on the principal diseases of the spinal cord,—those with which you are most likely to meet in practice. There may be a congested or hyperæmic state of the cord, or it may be anæmic, just as we had hyperæmia and anæmia of the brain. As in the brain we sometimes have an inflammation of its substance, or of its coverings, so we sometimes have an inflammation of the substance of the spinal cord, or of its enveloping membranes. In the one case we have cerebral meningitis; in the other, *spinal meningitis*. Again, we have encephalitis, and corresponding to it is the inflammation of the substance of the cord itself, which we call *myelitis*. Not only does it behoove you, as medical practitioners, to be able to distinguish *myelitis* from *spinal meningitis*, but you should have a good idea of the symptomatology, prognosis, and treatment of all diseases of the cord. The first disease that I shall take up to-night is *spinal meningitis*, which is, as I have just told you, an inflammation of the membranes enveloping the spinal cord. You recollect that in cerebral meningitis the inflammation is generally located in the internal membranes; and the same occurs in the disease we are now considering, the membranes inflamed being the pia mater and the arachnoid. The dura mater is sometimes, though rarely, involved. Males appear to suffer more than females. The disease is most common between the second and seventh years in children, and between the twentieth and twenty-fifth in adults.

The *causes* generally leading to the production of spinal meningitis are injuries, blows, concussions, and exposure to cold (especially in persons of a rheumatic diathesis); besides these, the rheumatic poison itself, as well as other toxic agents in the blood, has a tendency to act as an exciting cause of this inflammation. The reason why it is of great importance to study the symptoms developed in spinal meningitis is, because rheumatic affections are very apt to be mistaken for this spinal disease. On this account you should be doubly on your guard in the diagnosis.

The ordinary grouping of *symptoms* in inflammation of the spinal meninges is very much the same as in cerebral meningitis, with one exception. This exception, and you will naturally expect it, is the absence of any manifestation pointing to a disturbance of the intellect, for we are not dealing here with the coverings of the brain. But in spinal as well as in cerebral meningitis a prominent symptom is violent *pain*. This pain presents certain peculiarities enabling you to recognize it, being of a shooting, darting character, generally situated in the neighborhood of the spine, though it darts in the direction of the four extremities. A marked condition of *hyperæsthesia* exists, forming also a prominent symptom of the disease. The pain is always aggravated by any muscular movement,—so much so that patients suffering from spinal meningitis have a *well-defined decubitus*. They commonly sink in the bed in such a manner as to avoid any motion, shrinking instinctively from changing their position, the slightest movement or pressure seeming to augment their sufferings, often rendering them intolerable. In addition to these symptoms, and ordinarily after the disease has existed for a certain length of time, a *muscular contraction* of a tetanic character, especially of the muscles of the back and neck, sometimes amounting to opisthotonos, is developed; this is usually so strikingly peculiar that in some works you will find plates representing this condition of deformity, which deformity is generally dependent upon the continued contraction of the voluntary muscles. This is especially the case in the chronic form of the affection. Still, we find more or less contraction in acute cases after the initiatory symptoms have lasted for some time. As I have stated, the pain is excessive and burning, and darts in the direction of the extremities. In the acute form it is always accompanied by a *high fever* and

insomnia. A difficulty in emptying the bladder supervenes, as also obstinate constipation, and at times the sphincter vesicæ is so affected as to require catheterization. This the patient dreads. His decubitus is characteristic, and there is a marked intolerance of interference, even on the part of the physician,—the patient being continually apprehensive of severe pain from the slightest movement. If the effusion is great, the paralysis will be correspondingly profound, and in all cases the loss of power extends gradually upwards. There is more or less dyspnoea in all instances, and a disagreeable constriction around the abdomen and lower part of the chest, of which the patient greatly complains. Great prostration, delirium, and coma supervene in fatal cases. Sometimes there is *priapism* (a persistent erection of the penis) of an obstinate and troublesome nature; this, you recollect, may occur in other spinal diseases. Since, in rheumatism, the pain is also intensified by any muscular motion, in making a diagnosis of the case, should you not be very careful in your inquiries and in the search for true spinal symptoms, you will, as is done every day, mistake a very serious disease for an inflammatory rheumatic affection,—so closely does the former simulate the latter affection. In a post-mortem examination, if we inquire into the condition of the spinal membranes, we shall find some very plain traces of the storm which has passed over the pia mater and the arachnoid; there will be an opacity of these membranes, especially of the latter, with an effusion of serum between them. At times we shall even find a collection of purulent matter; and, in rigid cases, softening. If the disease has been severe, and serous effusion has taken place from the over-congested state of the spinal meninges, we shall have had a paraplegia during the patient's life,—that is, a paralytic condition of the lower extremities. However, this paraplegia is usually slight, incomplete, and poorly defined, unless the effusion has been decidedly great, which is rare. Undoubtedly the motor affection is far less determined than in myelitis.

The *prognosis* of spinal meningitis is always grave. This is the case in any inflammatory condition of the meninges, whether cerebral, spinal, or a combination of both. Still, spinal meningitis is sometimes amenable to prompt and energetic treatment. The most generally successful treatment is the application of counter-

irritants, warm poultices, pustulation, and cupping all along the spine. Then the free administration of iodide of potassium, or of bromide of potassium, ergot, and belladonna, often seems to have a remarkable effect in controlling the excessive accumulation of blood in the substance of the spinal cord, or in its membranes. As we come to study these several diseases more thoroughly, we shall find that we can reduce their pathology to two propositions,—two abnormal conditions,—namely, a hyperæmia and an anæmia; and just in proportion as the symptoms evince a hyperæmic or an anæmic condition of the cord must the remedies employed be diametrically opposite in their effects. This important fact is not always realized by practitioners, and the result is often very unfortunate. Some physicians use strychnia indiscriminately in these cases, believing that it has a tendency to relieve all spinal troubles; but I have already told you that strychnia increases the polarity of the cord, rendering it more hyperæmic than it already was; and, therefore, whenever a condition of irritation, or a state of hyperæmia of the spinal cord or of its membranes, exists, the use of strychnia must be positively avoided as deleterious in its effects.

The different remedies used in spinal diseases can be grouped, according to the experiments of Brown-Séquard, as follows: strychnia for increasing the circulation of blood in the spinal cord (quinine and iron being less energetic remedies for accomplishing the same purpose); ergot, belladonna, and perhaps bromide of potassium, for diminishing the circulation. Hence, if the disease be the result of a hyperæmic condition, give belladonna, ergot, etc.; on the other hand, should an anæmic condition exist and you desire to promote an afflux of blood, give strychnia. Of the efficacy of these different remedies there can be but little doubt; but dissimilar remedies generally produce dissimilar effects,—the same remedies often having different results on diverse portions of the body. So belladonna and ergot both act upon the unstriated muscular fibres, but each with preference on fibres in different localities,—belladonna, for instance, having a greater action on the muscular fibres of the iris, thereby dilating the pupil: therefore, if it be our desire to produce a muscular relaxation of the iris in those parts, to control a state of inflammation, we use belladonna or its alkaloid. Again, if we wish to produce a stimulation of the unstriated muscular fibres controlling the blood-vessels of the

mammary glands, thereby diminishing the state of hyperæmia and checking the secretion of milk, belladonna is an invaluable agent, not having its equal. Or if there is an irritable condition of the bladder or its *bas-fond*, and we find it necessary to regulate the circulation, we again give belladonna, which, by contracting the blood-vessels in those parts, produces the desired result. It is on account of this property that belladonna is frequently used in the treatment of incontinence of urine. But if it be our aim to act on the uterus, or to single out the blood-vessels of the cord,—or rather the muscular fibres controlling the arterial circulation in the meninges or the substance of the medulla spinalis,—we select ergot, finding that, just as belladonna exerts a more direct action on the arteries of the retina, the mammary glands, or the bladder, ergot possesses a greater affinity for the vessels of the uterus and of the cord. Therefore the choice in these remedies must be governed by the localities on which they are desired principally to act, notwithstanding their mode of action appears to be identical. But what I most ardently desire to indoctrinate you with is the necessity of asking yourselves, whenever called upon to treat an affection of the spinal cord, Have we here an excess or an insufficiency of blood? If an excess, have resort to ergot, belladonna, bromide of potassium, etc.; if an insufficiency, endeavor to remedy the pathological condition by the use of strychnia, or brucia, quinine, and iron.

So much for spinal meningitis; and I trust that you now have a good general idea of the disease. The next form of spinal affections to be considered is *myelitis*. By myelitis is meant an inflammation of the substance of the cord, from *μυελος*, marrow, *plus* the terminal *itis*. I shall try to put the differential diagnosis between this and the preceding disease in a plain and concise form, but shall first speak of the symptomatology. Myelitis being an inflammation of the substance of the cord, it follows, as a matter of course, that the symptoms will be serious and the results disastrous in proportion to the importance of the part affected and the severity and duration of the attack: this is more apt to be the case than in spinal meningitis. Myelitis is not marked by any uniform set of symptoms. Although in myelitis there is considerable pain in the neighborhood of the affected portion of the cord, it is less severe than in meningitis. This pain may be situ-

ated in the cranial prolongation of the cord, or in the cervical, dorsal, or lumbar region. It is increased by the application of heat or of cold (as of a sponge dipped in hot water or in ice-water), or by pressure. As I have said, the symptoms developed from the inflammation are intense in proportion to the importance of the part involved. Hence, if the inflammatory condition be in the intra-cranial portion of the cord, the symptoms will be manifest and the results very disastrous. From your anatomical knowledge you will understand that the functions of deglutition, articulation, and respiration must be seriously implicated where the inflammation is intra-cranial. There will also be deep-seated headache, convulsive movements of the face and head, and trismus. The pulse and cardiac action will be irregular. The life of the individual is then in great danger, the disease almost invariably proving fatal.

If the disease be cervical, the result will be serious in proportion as the affected part is situated high up or low down. If the inflammation of the cord be above the origin of the phrenic nerves,—dipping into the substance and involving the deep layers,—it follows that there can no longer be any innervation of the diaphragm, and the patient is certain to perish. But should the inflammatory condition be situated in the *cervical* part of the cord, and below the origin of the phrenic nerves, the symptoms will be acute pain in the nucha, great dyspnœa, impossibility of elevating the head, and paralysis of the upper extremities. The organs of deglutition and of articulation will also be involved.

If the dorsal and dorso-lumbar parts be affected (and this, by the way, is what you most generally find), we expect, of course, to find formication in the fingers and toes, and convulsive movements of the trunk. We find symptoms of an involvement of the intercostals and sphincters, paralytic phenomena of the urinary and anal sphincters, and also a marked *paraplegia*, or paralysis of the lower extremities (existing particularly when the lumbar portion is attacked, as is most commonly the case), in contradistinction to paralysis of the upper limbs, when the cervico-dorsal parts are those which are affected. The feeling of constriction—as of a cord tied tightly around the abdomen—is more common in this lumbar form. Now, in proportion to the part of the cord affected and its particular structures, do we find a development of symptoms of a morbid derangement in

those organs which depend for the natural performance of their functions upon the healthy condition of the cord. This being the case in all forms of myelitis, there is more or less involvement of its gray matter, which pathological condition produces two symptoms dependent thereupon. *First*, if the paraplegia be not completely developed, a loss of co-ordination of muscular movements in some cases is more or less evident. Of course, if the paraplegia be sufficiently marked, this lack of co-ordination will consequently not be evident. But since the inflammation which attacks the spinal substance does not proceed rapidly, but generally by gradual invasion, the development of the motor paralysis will, *pari passu*, be gradual with the progress of the former. We have, therefore, as a never-absent symptom, more or less complete paraplegia. In myelitis you may always expect paralysis of the sphincters, which is an important differential symptom from reflex paraplegia, in which the same condition does not occur. Then in proportion as the paralysis of the lower extremities is complete will you have more or less atrophy of the voluntary muscles, both from want of use and from more or less involvement in the lesion of the trophic nerve-cells. They are no longer under the control of the will, and as the affection advances there will be a fatty degeneration of their ultimate fasciculi, and they will no longer respond to the most powerful electric currents. Again, we frequently have ulceration and sloughing of the nates or buttocks, or of any part that is continually subjected to pressure: this is quite characteristic of myelitis. The gradual abolition of reflex movements in the palsied limbs is the *second* result of lesion of gray matter presiding over their nutrition, and is an important symptom. Jaccoud, however, believes exaggerated reflex power in paralyzed limbs in general, and spinal epilepsy (a combination of clonic and tonic spasms affecting paralyzed parts in certain affections of the spinal cord) in particular, to be a positive sign of organic paraplegia. In addition to the above symptoms, there is always present an *alkaline decomposition of the urine* within the bladder, generally leading to very distressing symptoms, a severe vesical irritation, with an accumulation of thick, stringy, ropy mucus, the urea being decomposed into carbonate of ammonium, causing a sort of chronic vesical catarrh, which is always a source of great annoyance to the patient. Anæsthesia is another

symptom which sooner or later will be developed and become complete.

The next question is, What is the pathological condition of the disease? In reply to this, I will say that it is a true inflammatory state, bearing a decided resemblance to encephalitis and its different grades. The myelitis may proceed to absolute softening or supuration; and embolism or thrombosis is not infrequently productive of softening of the spinal marrow. You will sometimes find it next to impossible to tell whether the softening depends upon this cause or upon a primary myelitis. Besides the softening, the inflammation of the cord may produce a condition of abscess, superficial and limited, or in the substance, as in encephalitis. Ramollissement is the most frequent result, which cannot be distinguished from non-inflammatory softening by the naked eye. Some portions are sometimes found indurated.

Now, what are the *causes* of myelitis? They are exactly the same causes as those of spinal meningitis, namely, severe blows, injuries, concussions, exposure to the vicissitudes of the weather, the existence in the blood of the rheumatic, syphilitic, or scrofulous poison, or of a *materia morbi* of any kind, as during the progress of fever. Care must be taken not to confound lumbago with the pain of myelitis.

As regards the *treatment*, iodide of potassium is the remedy most extolled, and in suspected syphilitic cases may be advantageously administered in enormous doses. Should you have a case of myelitis in its incipient or inflammatory period, and recognize and treat the disease before the supervention of softening, you may accomplish great good by the administration of those remedies which are best calculated to diminish the circulation of blood in the spinal cord.

The third form of spinal disease of which I shall speak is *hemorrhage in the cord*. We have already studied the mode in which hemorrhage of the brain, or of its coverings, is brought about,—by the rupture of an artery, for instance, owing to a diseased condition of the vessel; and so it is in hemorrhage into the spinal cord: the extravasation is produced by the same causes, though probably more often by caries of the spinal bones, dislocations, recent deformities, or accidents. The rupture of the vessels by the pressure of the bones upon the cord, a straining from

too great muscular exertion, or a severe blow, may give rise to a hemorrhage into the substance of the cord, or its coverings, or between the vertebræ and the dura mater; though it most commonly occurs between the membranes, rarely into the substance, and still more rarely outside of the dura mater. Here, as in myelitis, the symptoms developed will vary according to the different parts in which the hemorrhage is situated, though the blood has always a tendency to gravitate towards the bottom of the canal. In these cases we always have a sudden, abrupt *paraplegia*, in contradistinction to the paraplegia of myelitis, which is gradual, slow, and often almost imperceptible. In hemorrhage, then, the paraplegia is sudden, and the only instance where it is not rapid is when the hemorrhage is limited to a circumscribed space within the substance of the cord, is slight, and remains *in situ*, unless a subsequent extensive rupture ensues, which will of course eventuate in profound and complete paraplegia. The phenomena of motor disturbances in hemorrhage, then, are, as a rule, rapidly produced and manifested.

Now, to recapitulate, in *spinal meningitis* we have hardly any paralysis at all; in *myelitis* it develops gradually, and increases in intensity as the disease becomes more and more hopeless; and in *hemorrhage* the paraplegia is very abrupt, constituting the initiatory phenomena, and generally the immediate result of some accident or other cause, evident to the physician. Several of you may remember a case we had last year of a man who, in lifting a barrel of pork, strained himself; he felt something giving way at the time, and paraplegia instantly supervened. Until his death, which took place a few months afterwards, he remained perfectly paralyzed. Several of the gentlemen diagnosticated the case at the bedside simply from the history of the accident.

Among the characteristics of spinal hemorrhage is a sudden pain in the back: this, of course, is readily traced to the cause of violence, whether a fall, blow, concussion, or otherwise. Or the history may lead us to ascertain the existence of a pathological condition of the blood-vessels, which would furnish ample evidence of the etiology,—as, for instance, a supposed *endarteritis deformans*, of which you have heard so much, which may have caused a giving way of these vessels: however, this occurrence is rather infrequent. Spinal hemorrhage is, as a matter of course, extremely

fatal. But what I wish once more to impress distinctly upon your minds as of great importance in the diagnosis, is the existence of pain, and a very *rapid supervention* of paraplegia. Severe convulsions often occur. There is no loss of consciousness in spinal hemorrhage.

As to treatment, use the ordinary remedies, although very little is to be accomplished; just as in cerebral hemorrhage, the mischief has already been accomplished before you see your patient; the clot is there, and you cannot get rid of it. If not fatal, it ultimately leads to tissue-changes,—to an inflammatory condition of the medullary substance itself; and it produces a secondary myelitis, in the same way as an encephalitis is frequently produced after cerebral hemorrhage. Cold applications and quiet may be resorted to should the patient survive.

I shall now, for the sake of your own convenience, group these diseases in such a way as to facilitate you in making a *differential diagnosis*. First, in *spinal meningitis* a characteristic symptom is a peculiar pain, increased on movement, which darts in the direction of the extremities, and is rheumatic in character. Muscular contractions, hyperæsthesia, and incomplete paraplegia are present. The production of a remarkable muscular deformity in chronic cases, from spastic contractions, and also a peculiar decubitus in acute cases, are characteristic.

In *myelitis* we find as prominent characteristic symptoms the invariable presence of alkaline urine, and the slow, gradual, and progressive development of complete paraplegia, which is more marked and definite as the disease advances and becomes hopeless. Complete anæsthesia, and gradual abolition of the reflex movements, are invariable concomitant developments. In *spinal meningitis* there is little paralysis; it is rarely marked, and is incomplete in character; in *myelitis* it is marked, and generally more profoundly affects the sphincters.

Again, *spinal hemorrhage* is recognized by the rapidity of advent of the paralytic phenomena, accompanied by intense pain in the back, and the abrupt appearance of the paraplegia, as opposed to its gradual supervention in *myelitis*. In fact, I know of no disease of the cord in which the paralytic manifestations are so sudden as in spinal hemorrhage, with the exception of hysterical paraplegia. However, the history of the case, and several characteristic features

of hysteria, which I fully discussed while speaking of hysterical palsy, will enable you to make a correct diagnosis.

In *congestion of the cord* there is a dull pain in the lower portions of the cord. The palsy generally progresses from below upward, and is always incomplete in character. The sphincters are not affected. The paralysis is preceded by a feeling of numbness, and it is worse in the morning, or whenever the patient has been for any length of time in the recumbent posture.

Besides the above forms, we have the possibility of *concussion of the spinal medulla*. Just as concussion of the brain may produce disastrous results, so the jarring of the delicate fibres of the cord may produce certain symptoms, which, if overlooked or not properly treated, may be the forerunners of a progressive and disastrous spinal disease. The concussion may often be slight, as from alighting heavily from a carriage, or any sudden jarring, and nothing may be felt but a disagreeable sensation of formication in the fingers and toes. The concussion not having been in the brain, there will be no cerebral symptoms, and the symptoms at first will be of an insignificant character,—the spinal cord being the great centre of involuntary action, not of the intellect. If, however, there has been any serious jarring, and you recognize certain spinal symptoms, keep the individual quiet and on his back; do not attempt to treat actively; seek by cautious measures to avert the impending storm, and the recovery may be rapid. Just as in the brain a concussion may develop an insidious and severe inflammatory action, so a serious form of myelitis may be the result of an apparently trivial concussion of the cord; and if this has not been properly cared for by the physician in the very beginning, some form of spinal disease may develop itself which will afflict the patient for life.

Having spoken of spinal diseases, I cannot dismiss the subject without referring to *spinal tumors*. If these develop, as they usually do, in some particular portion of the cord, the symptoms will depend upon the anatomical situation of the part affected; and as generally the tumors are either of a cancerous, aneurismal, syphilitic, hydatid, serofulous, or exostotic nature, so will the general condition point to a cancerous, syphilitic, or serofulous cachexia. Again, as there is more or less encroachment on the spinal medulla will there be more or less severe symptoms of neu-

ralgia of the cord, or violent spinal pain, accompanied by cramps and convulsive movements; and as the pressure is limited in character so will the resulting symptoms be limited. The anæsthesia, hyperæsthesia, or the spasms, are dependent upon certain parts of the cord involved, and they may affect one or more muscles, or a group of muscles, according to the region of the cord diseased. The paralysis of motion always precedes that of sensation.

The last form of spinal disease left for consideration, and one concerning which you will hear much and read a great deal, is the so-called *spinal irritation*. Many physicians attach great importance to certain nervous troubles, which, not being able to explain otherwise, they refer to a spinal irritation. If they do not understand certain symptoms, they commence pounding the spinal column, under the supposition that upon reaching the affected part they will there find a certain hyperæsthesia, and a pain on manipulation, which will cause the patient to wince. It frequently happens that, in such an examination, the patient becomes tired or restless, and, not desiring to have this performance continued, perhaps winces. The doctor at once jumps at a conclusion, exclaiming, "A plain case of spinal irritation!" In former times, all such affections were considered congestive in character; the modern tendency is to ascribe most of them to anæmia. Although there is no doubt that in some obscure nervous affections we may really have a spinal irritation, there nevertheless will exist a profound general anæmic condition in many of these cases, of which it is the underlying pathological state, and usually an active cause of a hysterical or a hypochondriacal condition; and often chloro-anæmia, with which it is so peculiarly apt to be associated, is superadded. These conditions are generally made manifest by some neuralgic trouble, from a want of red corpuscles in the blood,—a sort of leucocythæmia, of which the pathological interpretation is, spinal irritation.

I grant that we usually have a condition of general anæmia which extends not only to the spinal cord, but also to the brain. It sometimes happens that a woman consults a physician, and he finds that she has an exhaustive leucorrhœal or menorrhagic discharge, or some other catamenial disturbance; she has mastodynia, pleurodynia, or intercostal neuralgia, the clavus hystericus,

or the globus hystericus, and the evidence of a hysterical constitution; her pallor is marked, and her general appearance decidedly anæmic; perhaps, upon being questioned, she will state that, in addition, she suffers from some gastric or dyspeptic disturbances,—the stomach not seeming to perform its regular duty properly. The doctor percusses the spine, and finally, reaching a tender vertebra, is, perhaps, a little rough in his manipulations. At all events, he hurts the patient, or she thinks she is hurt, and the diagnosis of spinal irritation is formed. But, gentlemen, even if all this be sanctioned by authorities, I must confess that I cannot understand these limited conditions of anæmia any more than I can those momentary congestions of the brain, which play so great a part in the diagnoses of many physicians. I have told you of the fleeting congestions which doctors so often confound with attacks of vertiginous epilepsy, etc., which latter, in fact, proceed from deeply-seated disturbances near the medulla oblongata. When, in the so-called spinal irritation, we have certain symptoms of nervous trouble, with a peculiar concurrence of conditions, the manner of manifestation of which is irregular, it is not always to the former state that we should ascribe them, neither to the spinal centres in general nor to spinal anæmia in particular, but to an anæmic condition of the entire system, which lies at the root of the whole difficulty.

Of course, if this doctrine be true, we ought to find the morbid condition relieved by good remedial agents of a constructive nature,—such as are employed to tone up the system,—certain blood-tonics, like quinine, or the ferruginous or arsenical preparations; and these, in point of fact, do have the most beneficial effects. In addition to these, the use of mild electric currents, and, above all, a highly nutritious diet and favorable hygienic condition, are the only available means for the successful treatment of this form of disease.

The inference is, that spinal irritation is nothing more than a general anæmia, the influence of which is exerted not only upon the nervous centres, but also upon all the tissues of the body.

LECTURE XXVIII.

PROGRESSIVE MUSCULAR ATROPHY.

Symptoms.—Parts first affected.—Absence of Systemic Disturbances.—Pathological Anatomy.—Characteristic Anatomical Alteration.

GENTLEMEN,—Mr. N. R., who has kindly consented to appear before you to-day, is afflicted with a most interesting form of disease, which it will not often be in your power to witness, owing to the rarity of its occurrence. Without a thorough acquaintance with the clinical features of the disease, it would be easy to overlook its existence in its earlier manifestations, or to mistake it for other affections. The case presented for your consideration being a typical one, it behooves us to study it with care and earnestness. This patient, whom Dr. Hodgen had the kindness to send me, is a young, vigorous man, nineteen years of age, well formed, and apparently of unusually fine muscular development. He is a coach-painter by trade, and has never been seriously ill, with the exception of a mild attack, several years ago, of what, from his description, I suppose was spinal meningitis. Since early childhood he has experienced a tremor of his extremities, which, though slight, is nevertheless, as you observe, quite apparent. This symptom is one of minor importance, as it seems almost to have been congenital, but in the diagnosis it is an element not to be overlooked.

The patient seeks medical advice for "a weakness of his hands;" and, upon careful examination, I find that the thenar and hypothenar eminences of both hands are atrophied, those of the right hand having entirely disappeared; the interossei muscles are much wasted, and in one or two places have entirely disappeared, leaving deep excavations in the palm of the hand in total atrophy. The extensors of the fingers are also perceptibly weaker than those of the left hand, but are too weak to produce paralysis.

by the atrophic changes in the muscles of the hand already mentioned, and prevents our patient from performing many of those nicer movements which require muscular precision. He cannot pick up small objects, tie his cravat, abduct or adduct his thumb and little finger on the right side, where the ravages of the disease are more extensive; yet there are no evidences of central disease, no cephalalgia, no psychical disturbance, no marked anæsthesia, no disturbances of any of the nerves of special sense. The motor impairment is, therefore, *peripheral* in its origin, whether it be due originally to nervous or to muscular lesions, a question which will occupy our attention farther on in this lecture. When we consider the occupation of this patient, coupled with the slight diminution of power in the extensors, a suspicion of lead palsy suggests itself; but the absence of a blue line on the gums, and the perfect retention of electro-muscular contractility in the muscles not completely atrophied, together with pathognomonic features of another disease which are manifest upon a careful examination, more than satisfy us that we have to deal not with lead palsy, but with progressive muscular atrophy.

I shall now rapidly enumerate some of the more important symptoms of this affection, in order that you may better appreciate this rare opportunity which presents itself for the study of an affection most interesting in its course and most important as to diagnosis, in order to alleviate a disease both progressive and obstinate, and which admits of few, very few means of palliation, not to say cure. We shall deem ourselves most fortunate if we can prevent its further progress, and shall abandon from the commencement all hopes of restoring tissues unalterably damaged. The atrophic mischief in the affected muscles, therefore, will give us little concern, as we know of no remedies which can reorganize tissues hopelessly destroyed. Now, this disease presents features so characteristic that, once observed, it can never be mistaken. The patient first complains of weakness,—of an increasing inability to perform certain muscular movements. This weakness is at first confined to one limb or to one muscle, or to a certain group of muscles in one limb, and sometimes a symptom manifests itself which is well marked in the gentleman whose case we are at present considering. I allude to the so-called *fibrillary contractions*, which are produced by vermicular move-

ments of certain fasciculi of the affected muscles, causing a sensation as if something were moving under the skin. Trousseau likens these movements to those of whip-cords tightened and relaxed under the skin. At times they may be unperceived, and when they do occur they are involuntary. The rapidity which characterizes these contractions is an important element in their recognition. The temperature of the affected parts is almost always lowered; and you may notice what cold, clammy hands our patient has.

So far, the disease has not attacked any other muscles than those in the palmar region. This, again, is a usual occurrence, as in most cases the invasion is slow and insidious and its first effects are strictly localized. Later, other muscles and groups of muscles may be invaded by the pathological process, if we cannot succeed in staying its onward progress. Yet even in its progression it presents certain well-marked symptoms, not to be overlooked, as it attacks the muscles *at random*, involving one here, and perhaps sparing one immediately adjacent to it; but in the more general varieties of the affection, all the voluntary muscles may become affected, reducing the unfortunate victim to a state of utter helplessness. After one limb has been attacked in this gradual manner, the opposite one may be involved, and in course of time the lower extremities are in turn implicated in the progressive atrophy: exceptionally and very rarely do they suffer first. In a case sent to me by my friend Prof. Boislignre last fall, the lower extremities were first involved. The tibialis anticus and peronei muscles on both sides had almost entirely disappeared, whilst the muscles of the thigh and upper extremities were intact. To realize fully the curious manner in which the muscles become diseased, you must bear in mind the fact that, whilst some fibres in a given muscle are atrophied, others in immediate apposition may remain intact; and this explains why the muscular contractility so long remains but little impaired, as contractions will ensue so long as there are healthy fibres to respond to the galvanic or faradic stimulus.

You will observe the characteristic emaciation in this patient; but do not fail to remark that this emaciation does not involve the whole right upper extremity, as is observed in paralysis of central origin, much less is the emaciation general, as in most

progressive diseases. This important diagnostic fact admits of an obvious pathological explanation, as there is shrinking and thinning only of the parts which correspond with the site of the atrophied muscles. The favorite seat of election of progressive muscular atrophy in commencing its work of destruction is peculiarly in the thenar and hypothenar eminences of the right hand. Dr. Duchenne has seen the first atrophic incursions in the muscles of the trunk, but such cases are the exceptions to the general rule. Next in order, the interossei, the flexors and extensors of the fingers, and sometimes the muscular masses of the posterior region of the fore-arm, become atrophied. Later, the muscles of the trunk and of the lower extremities are attacked. These muscular organic changes prevent the proper co-ordination of the affected muscles, and as the antagonistic movements of muscles not diseased are still exercised, characteristic deformities will not fail to be observed. You see in this case how, notwithstanding the startling atrophy of the muscles of the hand, those of the fore-arm are plump, firm, and prominent, indicating a potential faculty which you would find it unpleasant to have exercised upon you should this gentleman become enraged.

In this disease, notwithstanding its advanced stage, the perfect condition of the organic functions, the absence of disturbances of the system, will be in remarkable contrast with the sad havoc among the muscles. The appetite remains good, and the digestion is perfectly regular. Towards the close of the malady, unless the patient dies of some intercurrent affection, he is reduced to the utmost extremity of uselessness and helplessness. The muscles of articulation and deglutition being impaired, speech is more or less impossible, and feeding the patient is attended with imminent risk of choking, the horrors of which are intensified by a rapidly-increasing danger of asphyxia from paralysis of the muscles of respiration, especially the intercostals; and yet, amid all this wreck, the intelligence remains active and unaffected. Trousseau cites the case of a lady who died in the last stage of the disease: "She could scarcely breathe, and no longer spoke; but her eyes still retained all their vivacity, and reflected her intelligence, which did not forsake her. She could still contract some of the muscles which supported the head, and those of the index-finger of her right hand. During the last days of her existence she

conversed with her children by means of this finger. Several sets of alphabetical letters, like counters, had been procured for her, and with her finger she put the letters together, composing words and sentences. By that means she was enabled to make her will." In a case I saw last summer with Prof. Robinson, an old man, in the last stage of the affection, preserved his intelligence until the end, although for many months he had been almost incapable of moving a muscle or of exercising any relations with the external world except by his speech, which was not totally abolished. The disease, as its name indicates, goes on from bad to worse. Although its duration may occupy many years, yet it is apt to prove progressive from the extension of the pathological changes, in spite of our best efforts to meet them. The hereditary tendency of progressive muscular atrophy is marked, and females are rarely attacked. Young, vigorous men of the working classes are more liable than others to become its victims.

Now, gentlemen, as regards the pathological anatomy of progressive muscular atrophy, all agree that there is a fatty degeneration of the ultimate muscular fasciculi affected. The question of the *priority* of lesion, as to whether the morbid processes first attack the muscular or the nervous system, has involved pathologists in a fierce and protracted discussion, which is perhaps not definitively settled as yet, different authors arriving at different conclusions. Before I tell you those which I have accepted, I shall briefly enumerate a few of these different opinions. Many maintain that the vaso-motor nerves first are disturbed, and, as a matter of course, the mal-nutrition of the affected muscles is thereby explained. Virchow thinks that the anterior columns of the spinal cord are atrophied. Lockhart Clarke found, in some cases which he examined, in the gray substance in those regions of the cord which supplied the wasted muscles, numerous patches of transparent granular degeneration, and even cavities in the central portions of the cord. Other pathologists exclude all nervous lesions, contending that the whole difficulty is in the muscular tissues proper. In consequence of the frequency with which an atrophy of the anterior roots of spinal nerves is found, many allege this atrophy to be the lesion which is the *fons et origo* of all subsequent pathological processes. Now, as in progressive muscular atrophy the fatty degeneration of the ultimate muscular cells is never found

wanting, or, at least, their atrophy is always apparent, and as there are autopsies on record where the anterior roots of spinal nerves were found healthy, we consider this a strong presumptive reason for rejecting the spinal solution of the difficulty. Then, again, if the lesion of the spinal roots is accepted as an explanation, why do we have the disease attacking muscles, and even muscular fasciculi, *at random*? Should we not rather, in such a pathological condition of atrophy, expect to find only muscles and groups of muscles affected which are supplied by such spinal nerves? But this we know is not the case, as in the patient who is the subject of this lecture only a few muscles are atrophied, while others supplied by the same nerves are in striking contrast, being preserved and splendidly nourished. Then, again, we can account for the atrophy of the anterior roots of spinal nerves by the well-known fact that the diminution of the accustomed stimuli along these nerves may occasion their atrophy, as, for instance, in amputations near the trunk, when the nerves leading from the part are frequently found atrophied high up into the spinal cord itself. Notwithstanding the fact of the absence of visceral complications, and also that the unstriated muscles of organic life almost invariably escape, many are inclined to look to the *great sympathetic nervous system* for the proper solution of the pathological riddle.

According to Jaccoud, independently of the motor and sensory nervous filaments composing spinal nerves, there are sympathetic or trophic filaments distributed to the muscles, which maintain and regulate their nutrition. The complete inactivity of an entire nerve results, therefore, in entire abolition of sensibility, motility, and nutritive efficiency (atrophy). In nerve-trunks the three separate and individual filaments are so intimately associated that they cannot be separately injured, and consequently an entire lesion of such nerves produces always three distinct effects, as just described. If, however, there were a point at which these different filaments could be separately injured, the effect would be simple instead of complex,—that is to say, according as there existed only an abolition of movement, or one of sensation, or exclusively a nutritive alteration (atrophy). There is, in point of fact, a region in which such an isolation can be accomplished: for the motor filaments it is found in the anterior spinal region, including the anterior spinal roots as far as the intervertebral ganglia; for the

sensory filaments it exists in the posterior portion of the spinal columns and in the posterior spinal roots. The trophic filaments can be isolated in all that part of their track which they pursue before being merged with the peripheral nerve-trunks, namely, the gray substance of the spinal marrow from whence they spring, the anterior and posterior spinal roots by means of which they pass out of the spinal axis, the anastomosing branches which bind the spinal marrow to the sympathetic, and, finally, the sympathetic cord itself, including the branches distributed therefrom.

In all these places an isolated lesion can be accomplished upon these particular filaments, necessarily resulting in a nutritive insufficiency; if, moreover, such a lesion were progressive and extensive, the great portion of the muscular system would become atrophied without a primary or accompanying paralysis of movement or sensation.

Such is precisely the clinical characteristic of progressive muscular atrophy, the anatomical and pathological condition of which is a lesion of the sympathetic or trophic system (Jaccoud).

The different nervous lesions, according to the observations of different authorities, vary somewhat, and may be grouped into lesions involving the anterior roots of spinal nerves, those involving the spinal marrow itself, and those involving the sympathetic ganglia and nerves.

In France, atrophy of the anterior gray cornua of the spinal cord is considered the common and constant anatomical characteristic, an hypothesis which I need hardly say is more than doubtful.

In conclusion, we may affirm that the characteristic pathological and anatomical alteration in progressive muscular atrophy is a lesion of the sympathetic nervous system. Acting upon this view, and knowing the utter uselessness of all drugs as remedies in this disease, we will place all our trust in the galvanization of the cervical sympathetic, hoping by this means, at least, to arrest the progress of this direful malady; and, in so doing, our views are corroborated by the weighty authority of Althaus and Meyer.

LECTURE XXIX.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

Divisions of Sclerosis.—Symptoms of Multiple Sclerosis.—Diagnosis.—Prognosis.—Treatment.

GENTLEMEN,—To-day I present you a case of unusual interest,—one which affords you an excellent opportunity to study a comparatively rare affection, not easily diagnosticated, unless you possess some familiarity with the different clinical varieties of sclerosis.

The patient, John Parle, aged forty-two years, has been an inmate of the City Hospital for the last ten months; he is a laborer by occupation, and a native of Ireland. His disease commenced three years ago. He had always enjoyed good health prior to his present illness, with the exception of several attacks of intermittent fever, which yielded to the ordinary treatment. About ten years ago he fell from a hand-car, receiving a slight concussion on his left side, but recovered in a few days and resumed his ordinary employment. Six years ago it was supposed that he had contracted syphilis, a small sore having made its appearance upon his glans penis. As he has never suffered from sore throat, alopecia, secondary eruptions, or glandular involvements, I am not inclined to accept the opinion of his previous medical attendant, that he was at one time a victim of constitutional syphilis.

Three years ago he first experienced the prodromata of his present attack. The first symptom observed was a heaviness of his feet and legs, pointing to evident disturbances of motility. He, however, continued his ordinary occupation, without suffering any pain, until about six weeks afterwards. The pain then complained of was first felt in the lumbar region, and was of a sharp, piercing character. It was only of about six hours' duration, and then disappeared. Two weeks afterwards the same symptoms

reappeared with the same intensity, lasting for the same length of time. A month later, lancinating pains of great violence situated in the spinal cord and shooting down the legs and into the feet greatly annoyed the patient. These pains lasted for twenty-four hours, but have never since recurred. Some days subsequently, whilst walking from his boarding-house, his limbs seemed suddenly to give way, and he was obliged to maintain himself erect by holding on to a fence, gradually and with difficulty reaching home. Since then he has never been able to walk except with great caution, and at present requires the assistance of a cane, or of surrounding objects, and sometimes of friendly help, in order to accomplish ordinary locomotion with any success.

About the time of these occurrences a spasmodic movement or jerking of his feet developed itself, followed by a tremulousness of the paralyzed parts. The tremor was produced when he attempted to walk or to perform any voluntary movements. Before these most marked and pathognomonic symptoms occurred, he had some trouble in controlling the passage of his urine and fæces; he was then entirely unconscious of the acts of defecation and urination. A careful review of the present condition of our patient reveals the presence of a partial paraplegia. When the tremor occurs, which is never the case when he is perfectly quiescent, he loses all power and falls to the floor. He now, however, becomes conscious when the urgent desires which precede the necessity of relieving his bladder are experienced, but he is then obliged to introduce his finger into his rectum and press upward, which enables him to urinate without trouble; but should he retain his urine too long, it passes away incontinently. He has only a moment's warning previous to defecation, and he can by no effort suspend or delay the operation for an instant. Sensation is slightly impaired, and for three years he has had no venereal appetite nor any erection of the penis. His general health is excellent, and all his thoracic and abdominal organs are in a perfectly normal condition.

The different forms of sclerosis of the diffused and multiple kind may, according to Hammond, be divided clinically,—first, into two cranial varieties: the multiple cerebral and the diffused cerebral. The spinal cord may be attacked by a sclerosis of its antero-lateral columns, the symptoms of which would plainly

point to disturbances of motility ; or by sclerosis of its posterior columns, accompanied by marked sensory impairment, with a striking want of co-ordination of muscular movements as a pathognomonic symptom. We have an intermediate variety between cranial and spinal sclerosis,—the subject of to-day's lecture, multiple cerebro-spinal sclerosis. Tremor is developed only when the disease involves the superior cerebro-spinal ganglia, and constitutes a most important manifestation.

Until within comparatively a very recent period, paralysis agitans, multiple cerebral sclerosis, and multiple cerebro-spinal sclerosis were confounded under the common name of paralysis agitans. The two former are organic diseases of the nervous system, the latter is a neurosis of functional origin. Diffused sclerosis consists in a morbid proliferation of the connective-tissue cells or neuroglia, at the expense of the ultimate constituents of the nervous elements, encroaching upon them and leading to their gradual atrophy. It is characterized by induration and disappearance of the ganglion-cells and nerve-fibres, and is not distinctly circumscribed except by the anatomical limits of the region affected.

Multiple sclerosis, on the other hand, involves several parts of the same ganglion, and consists of nodules or plates of sclerosed tissue scattered throughout its substance. We are indebted to the comparatively recent researches of Messrs. Charcot and Vulpian for the proper elucidation of this difficult subject, and Dr. Meredith Clymer, of New York, was the first to present their views, somewhat modified by his own opinions, to the American medical profession. The initial symptoms of multiple cerebro-spinal sclerosis depend for their character upon the location of the first pathological changes, whether they occur in the brain or in the spinal cord. In the former case cerebral, in the latter spinal, symptoms will immediately attract our attention.

In the case under consideration the symptoms were *ab initio* distinctly of spinal origin. The ordinary cerebral symptoms are vertigo, epileptiform attacks, and derangements of the nerves of special sense, and *especially tremor preceding paralysis*, none of which have marked the progress of the affection in the patient I present to you this morning. Tremor itself is but an evidence of debility or nervous asthenia, and acquires significance according to

its clinical manifestations. When it *precedes* paralysis it points to multiple cerebral sclerosis, and is provoked by movements both voluntary and involuntary on the part of the patient; when it *follows* paralysis it is an accompaniment of the multiple cerebro-spinal variety of the disease, and is only developed by movements distinctly voluntary in their origin; it is often, as an isolated symptom, an evidence of commencing cerebral disease. Hence upon these last-mentioned facts the diagnosis of this case is based.

The tremor, nystagmus, festination, and a peculiar trembling of the tongue, without being pathognomonic symptoms, are oftentimes present, and are therefore truly significant. My experience does not lead me to believe that the sensory disturbances are very manifest, though frequently some slight sensory impairment exists; especially at times will we discover an absence of that exact "topographical knowledge" so familiar in the history of locomotor ataxia. Of the etiology of this affection I think it can be safely asserted that we know absolutely nothing, as it is shrouded in a mysterious darkness as yet unexplored.

In a diagnostic point of view it is important to bear in mind the following distinctions. *Paralysis agitans* is a disease of the nervous system dependent on a morbid condition of motor roots, in which there are no head-symptoms, the tremor is the initial phenomenon, and is continual. In *multiple cerebral sclerosis* tremor always precedes paralysis, is accompanied by festination, and the tremor is produced by movements, whether voluntary or involuntary. In *multiple cerebro-spinal sclerosis* paralysis precedes tremor, and the latter is only developed during voluntary muscular movements; it is likewise accompanied by festination.

In sclerosis of the *antero-lateral columns* there is no tremor, no festination.

The prognosis is the same as that of all progressive affections: we cannot possibly arrest them, and despite our best efforts they have but one termination.

As regards treatment, I have, therefore, little to claim your attention; for my faith in the efficacy of all remedies is weak, and beyond the judicious use of the continuous galvanic current applied to the brain, spinal cord, and sympathetic nerve, I think all recommendations useless and simply empirical.

LECTURE XXX.

BELL'S PALSY.

Pathology. — Causes. — Symptoms. — Prognosis. — Diagnosis. — Treatment.—Double Facial Palsy.

GENTLEMEN,—I desire to make a few remarks in regard to a very interesting form of paralysis which you will often meet with, and whose diagnosis is always of importance, as there is danger of mistaking it for a graver affection. I allude to a paralysis of the portio dura of the seventh pair of nerves, a disease which is commonly known as *Bell's palsy* (from its having been first described by Sir Charles Bell) or *facial palsy*. From your studies of anatomy you all know that the portio dura of the seventh pair supplies nearly all the facial muscles, and that it is in consequence of their innervation by this nerve that they contract, and give rise to the different expressions of the features indicative of the emotions. In fact, it is to this nerve that the human countenance owes its expression and the peculiar animation by which it is characterized. Now, this particular division of the seventh pair, the portio dura, as it is called, was formerly supposed to be a nerve of sensation, and in several cases of neuralgia it was considered that its filaments were irritated, and, consequently, it was sometimes divided. However, more recent physiological experiments and more thorough anatomical investigations have conclusively proven that this nerve is purely one of motion, and is not accompanied by any sensory filaments whatever; therefore, if its physiological action be interfered with, either traumatically or idiopathically, there will be a paralysis of those muscles which it supplies. Its physiological duty is to enable the different facial muscles to contract in such a manner as to represent faithfully the phenomena of emotions in the human countenance.

It is a matter of considerable importance to be able to recognize the several forms of paralysis of this nerve and their accom-

panying symptoms. The disease is generally peripheral in its origin; that is, it originates in a disturbance of the peripheral parts of the portio dura, central causes rarely leading to a paralysis of this nerve. In Bell's palsy, the origin, therefore, is not frequently central or cerebral, but is purely peripheral, and the disease consists usually in a modification of the physiological action of the peripheral distributions of the portio dura. I presume all of you will readily appreciate the difference between a peripheral and a central nervous disease: if the paralysis proceeds from central causes, its manifestations depend upon some deep-seated or organic disease of the brain or of the spinal cord, or upon some involvement of the nerve-centres proper; but when the disease is peripheral, it is not occasioned by any such pathological conditions: it is simply the peripheral part of the nerve which is involved and from which the paralysis is developed,—which is the case in most instances of Bell's palsy.

Before I begin to enumerate the different manners in which this paralysis may be evidenced, I wish to recall to your minds the origin and the course of the nerve whose abnormal state forms the basis of the ailment we are now considering. The seventh pair of nerves emerges from the posterior part of the pons varolii, and also from the lateral tracts of the medulla oblongata. It is even held by some authors that some of its fibres originate in the lateral columns of the cord, and that its deep fibres may be traced to the floor of the fourth ventricle.

Now, in a case of paralysis implicating this nerve, the pathological condition giving rise to the paralytic phenomena may be situated in any one of three different places. You know that after the nerve emerges from the medulla it enters the petrous portion of the temporal bone by the meatus auditorius internus; it then courses through the aquæductus Fallopii, and finally emerges from the bone through the stylo-mastoid foramen; here it divides, and sends filaments to the different muscles which it controls. We can divide the nerve into three distinct portions, namely, one portion entirely intra-cranial, between the points of origin and its entrance into the petrous portion of the temporal bone; one intra-osseous part, lying in the substance of the bone itself; and an extra-cranial, or muscular portion,—that portion of the nerve which has passed out of the stylo-mastoid foramen.

Any interference with or disturbance of this portio dura of the seventh pair of nerves, at any portion of its course, will necessarily lead to a paralysis of the facial muscles. Now, in the majority of cases the source of disturbance, the particular influence exerted on the nerve, exists in that portion of the nerve which has passed out of the stylo-mastoid foramen. Sometimes the nerve is affected while still in the petrous portion of the temporal bone, by caries, or by a disorganization of the internal ear, as by syphilitic disease of the bones, etc.; or it may be affected in its first portion—the portion included between its origin and the internal meatus—by a distinct set of causes, such as the pressure from an intra-cranial tumor, a disease at the base of the brain, or an affection of the cranial portion of the base of the skull, such as an exostosis, or an adventitious process of some kind. In all such cases symptoms of a paralytic character will follow. Again, after having made its exit from the skull, the nerve may be pressed upon by a tumor of the parotid, or by a swelling of the gland itself, causing a facial paralysis; or such a paralysis may be produced by the pressure of the forceps in protracted labor, and also—and not uncommonly—by blood-poisoning of a rheumatic or of a syphilitic character, especially the latter. Or the disease may be induced by exposure to cold, resulting in an inflammatory condition of the neurilemma, or those parts which surround the nerve; an exudation may take place, and by its pressure lead to a paralysis of the peripheral portion. In point of fact you will find that Bell's palsy sometimes distinctly originates in this manner. I remember the case of a lady who was traveling in a railroad car and was sitting near a raised window with one part of her face continually exposed to a draught; she caught cold, and a paralysis of the portio dura of the seventh pair was the result. Again, it has been found that any emotional disturbance may produce the disease; and Trousseau relates the case of a woman, in a hospital in Paris, who was so frightened by a severe thunder-storm that she was immediately seized with an attack of Bell's palsy.

We have now seen that violent emotions, exposure to cold, rheumatic or syphilitic blood-poisoning, swelling of the parotid gland, the use of the forceps in labor, caries of the petrous portion of the temporal bone, pressure on the intra-cranial portion of the

nerve by tumors, exostosis, etc., are some of the leading factors concerned in the production of Bell's palsy. But in point of fact, and as a general thing, the paralysis is idiopathic in character, affecting the peripheral portions of the nerve only, and that part, therefore, which has emerged from the stylo-mastoid foramen.

The next question is, How are we to recognize this disease? Provided we know the nature of the affection we are dealing with, relief is generally easily afforded. It is highly necessary to make a correct diagnosis, for if you should be called upon to treat a lady with a distorted countenance, and were to ascribe the deformity to an incurable pathological condition, and then a neighboring physician were to succeed you in the case and in a short time restore the patient, your reputation would be very seriously compromised. But if you bear in mind the anatomy of the nerve, and its physiological functions, you will readily understand that the detection of the symptoms during life must be easy; for the way in which we recognize most nervous diseases is by their anatomical relations, and the parts therewith associated; as also by the physiological formations of the organs affected, since there must necessarily be a disturbance or alteration of their functions. For instance, when there is an inflammation of the cortical portion of the cerebral convolutions, we will distinguish the condition by the mental actions of the individual; if there be a softening of the gray matter of the cord, we of course expect certain sensory disturbances, or abnormal changes in co-ordination; and where a paralysis of the portio dura of the seventh pair exists, we naturally examine the countenance, as it is in the muscles of the face that we expect to find the manifestations of the paralytic phenomena.

This being the case, I shall enumerate the different paralytic symptoms as they occur, from above downwards. First, we have paralysis of the orbicularis palpebrarum muscle,—the muscle of the eyelid. It is in consequence of the contractility of this muscle that we are enabled to close the eye. This muscle is supplied by the supra-orbital branch of the portio dura, from which it receives its innervation; consequently, in Bell's palsy we *always* find a disturbance of its physiological action.

Now, before going any further, I may state that, in the vast majority of cases, Bell's palsy is a unilateral disease; that is, it affects one side of the face only. It is, therefore, on the affected

side that the individual is unable to close his eye. Have you ever reflected what a serious matter it would be if you were no longer able to close your eyes? Such a condition would undoubtedly result in great mischief. In the first place, you would be deprived of the power of winking; the eyelids could no longer be brought together in quick apposition, and as a consequence the eyeball would no longer be lubricated. This involuntary winking, which is much quicker than thought (for it does not require any mental regulatory influence, but is due entirely to reflex action), subserves an important physiological purpose. And in what manner? You know that there are constantly floating in the air little particles of dust, finely-divided portions of matter, which are brought in contact with the eyeball, and are removed by the lubrication during the act of winking. You can readily conceive that if these particles of dust were allowed to remain and accumulate in the eye, they would necessarily act as irritants, and lead to a conjunctivitis or a chemosis, or even to ulceration of the cornea. This has actually happened; and persons having Bell's palsy are continually subjected to these accidents; but they generally obviate the difficulty by the use of the fingers, or by holding something to the eye in order to protect it.

Then, again, we have other disturbances, such as epiphora, or a continual flow of tears down the cheeks. This is due to the fact that the lower lid has also lost its tonicity; its inferior margin droops, and no longer directs the tears towards their proper channel, but allows them to course down the cheek, irritating its cutaneous surfaces, with very disagreeable results.

The act of winking is quite complex: it consists of three distinct sets of muscular contractions, under the co-operation of three different nerves. First, when the eye is irritated, a certain excitomotor irritation is put in action along some of the fibres of the first branch of the fifth pair, producing a necessity of winking, and this instinctive desire to wink is carried by sensory filaments of the fifth nerve. This influence reaches the pons varolii, and immediately a mandate—a new influence—is sent along the electrical wires of the portio dura to the orbicularis palpebrarum muscle to contract, which it does, and the eye rapidly closes. Now, if you were to wink, and then not be able to open your eye, your situation would be lamentable. This is exactly what

would happen were it not for a third nervous influence, this time propagated along another nerve and to another muscle, namely, by the oculi-motor nerve to the levator palpebræ muscle, directing it to contract, which it does, thereby opening the eye and completing the process of winking.

I have now explained the physiological action of winking,—a most important feature, as this operation is always interfered with in paralysis of the portio dura. The patient not being able to close his eye, it remains unduly open, and you can notice a wide separation of the eyelids, with a peculiar glaring or staring expression of the eye.

The reason I lay so much stress upon this feature is because I wish you all to remember it, as it is the *unequivocal characteristic symptom*—always present—in Bell's palsy; exactly as is the pathognomonic symptom of epilepsy,—loss of consciousness.

Another paralyzed muscle is the orbicularis oris, the muscle of the mouth. Of course, only one-half of this muscle is affected; and, from the want of tonicity and the condition of relaxation, the labial commissure is lowered on the affected side, and, as a consequence, there is a flow of saliva over the corner of the mouth. Besides this, you will notice that the mouth is drawn unduly towards the healthy side; the healthy muscles, being no longer antagonized in their actions, draw the face to one side. This is not all: every muscle of the face is involved in the paralysis, with the exception of some of the muscles of mastication, the masseter, temporal, and pterygoid muscles, which receive their innervation from the third branch of the fifth pair, which is partly a motor nerve.

Among the muscles supplied by the facial nerve is the buccinator. You undoubtedly know that chewing is a very important function as regards the preliminary changes which solid food must undergo before being carried to the stomach. The object of the buccinator is not only to preserve the rotundity of the face, but also to aid in mastication. When a person is suffering from an attack of Bell's palsy, the rotundity of the face is no longer maintained, and this muscle hangs loosely down, giving that part of the cheek a flap-sail appearance; but this does not prevent him from eating, for he has still the pterygoid, temporal, and masseter muscles left. The alimentary bolus, however, can no longer be

directed from one side of the mouth to the other, in order to be lubricated and acted upon by the saliva, which forms an important part in digestion; and if the bolus gets on the paralyzed side, how can it be propelled to the other? This must be a very annoying occurrence, and, if you should be at a meal with ladies, eating some soft substance, like mush, or bread and molasses, and have your buccinators suddenly fail in the performance of their duties, and be forced to have recourse to your fingers, you would be subjected to great mortification. This is exactly what occurs in Bell's palsy: the food is no longer propelled under the grinders on the affected side, but lodges between them and the cheek.

We have already seen that the patient cannot wink; he cannot shut his eye unless he uses his fingers; the saliva runs from his mouth; his buccinator fails to act. But is this all? No; especially if he be a romantic youth, and desires to depict the emotions of love. Let us suppose the right side to be the one paralyzed: he may attempt to smile, but his countenance on that side will be a blank while all is animation on the other. Neither could he whistle or blow out a candle. In Bell's palsy, the human countenance, the mirror of the soul, is rendered incapable of indicating the play of emotions, and can no longer convey them to the external world. How do we judge of a man's character? is it not always by his countenance? How do we know what is going on within him? is it not by his features? It is the contraction of the muscles of expression that tells us when a person is angry; they portray the feelings, affections, and sentiments, and indicate to others when a person is elated, or when in pain. In paralysis of the portio dura the side of the face affected is immovable, a blank; the face is expressionless, and there is no evidence whatever of emotion. You must see this condition in order to realize it and appreciate its peculiarities. Sometimes there is a difficulty in the pronunciation of the labials, and the articulation becomes indistinct. Add to all the above symptoms a paralysis of the *alæ nasi*, in consequence of which one nostril appears more or less closed and the other abnormally dilated, and you have the features constituting Bell's palsy.

These symptoms are all very important; and if you doubt it, you will be convinced when you are called upon to treat women in this condition, who are usually greatly distressed on account

of any disease that affects their appearance. They can no longer frown, laugh, or cry, at least on one side; their corrugator supercillii and half of the occipito-frontalis will be inactive, and, as a German authority remarks, there is no better cosmetic than Bell's palsy, the skin on the affected side being as smooth as alabaster. Now, recollect this group of symptoms, for you will surely meet it; and, as general practitioners, you are compelled to treat such diseases; you cannot send them to specialists. If the disease be not exceptionally obstinate in character, or due to a serious affection of the cranial bones, which is rare, and if it be simply peripheral and in the muscular portion of the nerve, the patient will recover rapidly, if your means of treatment are efficient. But if you display a want of confidence in your abilities, or an ignorance of the disease, or make a mistake in the diagnosis, giving at the same time an unfavorable prognosis, the patient will not employ you long, but will send for another physician, and, if he is successful, it cannot but be to you a source of great humiliation and mortification.

The *prognosis* depends entirely upon the portion of the nerve involved, and the cause of the paralysis. If the seat of the disease be intra-cranial, or within the petrous portion of the temporal bone, the prognosis is necessarily unfavorable, and the paralysis will probably remain for life. But if the disease be seated in the part of the nerve that has passed out of the stylo-mastoid foramen, it is easily cured; and this is what happens in eight cases out of ten. Hence the particular seat of the affection and its cause are two important subjects; and, in regard to the etiology, let me caution you that, if the paralysis be due to a syphilitic or a rheumatic blood-poison, anti-syphilitic or anti-rheumatic remedies are the only ones that produce beneficial effects.

In the *diagnosis* is there anything that this disease might be mistaken for? is a question replete with interest and importance. You may ask, Well, how is it when the affection is central in origin? Recollect that, as a rule, it is peripheral; but where there is a central brain-lesion, as, for instance, a clot, you will have more or less facial paralysis, or more or less involvement of the portio dura of the seventh pair. You will now probably think we are getting deeper and deeper into difficulty, and that, instead of making matters clearer for you, I am making them

more complicated. But I shall make them, I trust, very easy and plain.

I have already stated that Bell's palsy is generally a peripheral affection, and is manifested by a paralysis of the facial muscles. I have also said that if the disease be central there is more or less involvement of the portio dura, and a consequent facial paralysis. Now, how are we to discriminate? Trousseau gives us a few peculiarities, to which, as he says, there are no exceptions.

In the first place, in true Bell's palsy there are no brain-symptoms, unless the disease be situated, as it is exceptionally, in the intra-cranial portion. If it have not involved any central parts, there will be no hemiplegia, not even a threatening. You recollect that by a threatening of hemiplegia is meant the warning of which I have spoken, which consists of a certain weakness and numbness in the hand or leg about to be paralyzed. This warning is absent in this case. But, independently of that, we have here a golden rule, which you should all recollect: it is, that in Bell's palsy there is invariably an unequivocal and pathognomonic symptom,—the paralysis of the orbicularis palpebrarum muscle. And if, in your visits to the hospital, you take the trouble to examine some of the old cases of hemiplegia the result of cerebral hemorrhage or of softening, you will see that, though there is more or less facial palsy, the paralysis of the orbicularis palpebrarum muscles, if it exist at all, is incomplete, and that by an effort these patients can always accomplish the closure of the eye; whereas in Bell's palsy the closure of the eye is an absolute impossibility. Now, here is the differential point, which you must recollect, even if you should forget everything else I have said upon the subject.

It has been contended that a limited hemorrhage may occur in the neighborhood of the pons varolii, and that this may be expressed only by a slight facial paralysis, without any serious paralytic or hemiplegic phenomena. No doubt a small effusion of blood in the fourth ventricle or near the pons varolii may give rise to a slight palsy of the facial muscles; but this occurrence is so rare as hardly to be taken into consideration in the diagnosis. In such cases there would probably be other brain-symptoms of some kind, whose presence you might ascertain by close scrutiny.

I have now spoken about everything but the treatment; and here I shall give you another golden rule,—one that will be of

value to those who are interested in the therapeutic use of electricity. In all peripheral forms of paralysis, as in true Bell's palsy, the contractile power of the muscular fibres under the electric stimulus is wonderfully diminished; while if the paralysis is cerebral in origin the contractility will scarcely be affected at all.

In the *treatment*, in the first place, never forget that you should ascertain the cause of the trouble, and if you have done this and can possibly remove it, do so. Sometimes it is difficult, or even impossible; for instance, when the paralysis has been caused by protracted pressure of the fist against the head while sleeping, in the same way as a paralysis of the arm is sometimes produced while sleeping in a chair with the arm overhanging it and the chair pressing on the brachial plexus. Sometimes the cause cannot be ascertained at all, as when the disease is strictly idiopathic in character; but when it depends upon a rheumatic or a syphilitic diathesis, the treatment, being directed to the relief of these conditions, is generally productive of good effects. As a general remedy in Bell's palsy, there is none better than strychnia. If there be a suspicion of the rheumatic or syphilitic diathesis, give iodide of potassium; and if you find decided evidences of the latter blood-poisoning, a combination of mercury with iodide of potassium will be highly efficacious. As regards certain local applications, such as blisters and counter-irritants, I have no faith. But the treatment *par excellence*, and in which I have great confidence, is the systematic use of electricity. In trying the electric currents during the first period of the disease, you will find that if you apply the faradic or induced current the muscles will not respond at all; whereas if you use the continuous galvanic current (which must be interrupted by slowly and regularly breaking the current) you will obtain a decided muscular contractility. But after having used the current for some time you will find that later the muscles will respond better to the induced than to the continuous current. Always commence, therefore, with the continuous current; apply the positive pole to the place where the nerve emerges from the cranium, and the cathode over the paralyzed muscles, and single them out as you proceed. By doing this you will frequently obtain marvelous results. The cure will greatly add to your reputation; for though these diseases are simple, they are just those that most physicians do not know how to deal

with. Therefore never forget the use of the galvanic current, as I have just described. Always be careful, however, not to use too violent a current; remember that its proximity to the brain may cause you to do serious damage, endangering the patient's life. The first muscle to recover will be the orbicularis palpebrarum, and the others will follow in succession.

One more fact before concluding this lecture. When consulting the authors, you will find allusions to what is termed "*double facial palsy*." This consists simply in the same conditions that I have described, only that they exist in such cases on both sides of the face at the same time; that is, you have an affection of both facial nerves at one and the same time. This is a rare disease, in which the countenance presents the appearance of a perfect mask, without the slightest expression. The treatment is like that of simple Bell's palsy in every respect.

LECTURE XXXI.

ORDINARY AND REFLEX PARAPLEGIA.

Paralysis from Spinal Congestion.—Priapism.—Bed-Sores.—Definition of Paraplegia.
—Ordinary Paraplegia.—Causes.—Reflex Motion.—Reflex Paraplegia.—Etiology.
—Brown-Séquard's Table.—Treatment.

GENTLEMEN,—While speaking of spinal diseases the other night, I omitted to dwell sufficiently upon an affection called *congestion of the spinal cord*. It is a disease not infrequently met with; I shall, however, merely allude to its more characteristic features, several of its symptoms resembling those of reflex paraplegia. In congestion of the cord there is a dull pain in the lumbar or sacral regions, the paralysis is always of a limited nature, fluctuating and more or less partial in character, never complete; and you will generally find that when you have a paraplegia depending upon spinal congestion, its intensity will diminish or vary according to the changes in position which the patient may assume; so that when he has been lying all night on his back, thereby favoring the gravitation of blood and increasing the congestion of the spinal centres, the paraplegia will be worse in the morning.

The palsy progresses slowly from below upward, and is preceded by numbness, but the nutrition of the muscles is not affected.

Again, in cases of spinal congestion there is very little sensory disturbance, no anæsthesia of any importance, and the motor affection is limited and insignificant, the paraplegia varying in intensity. Disturbances of the abdominal circulation and alterations in the blood are the usual causes. Paralysis of the sphincters is absent.

You have heard me frequently speak of *paraplegia* of late, but before saying any more about it I must state that I omitted to tell you that in some forms of spinal disease, as in myelitis, there is generally a sensation of a cord tied around the abdomen, and when the spinal affection depends upon a hyperæmic state with an irritation of some motor nerves, more or less convulsive twitch-

ing and contractions occur; there is also priapism in these cases. Again, if any sensory disturbances follow the condition of excess of blood in the cord, there will be different forms of analgesia, or anæsthesia. Then, besides these phenomena, you also have certain peculiarities as regards temperature: there is a morbid, unpleasant sensation of cold.

Whenever there is too much blood in the cord or in the sympathetic ganglia (and these are of course always more or less involved), you find certain symptoms pointing to deleterious changes of nutrition in the condition of the patient. You have more or less irritation of the vaso-motor nerves, and, as a consequence, there is a great tendency to the formation of bed-sores. Now, how are these sores brought about? I have once before told you that when the vaso-motor nerves are irritated, these nerves will keep the arterioles in a state of contraction; of course there will be a disturbance in the circulation, the parts which are deprived of their usual nutritive supply begin to mortify and to slough, and, as this is more likely to occur in those portions of the body subject to constant pressure, bed-sores are generally the result. Besides this, the morbid innervation extends to the mucous membrane of the bladder, and the urine decomposes and becomes alkaline.

Now, by *paraplegia* is meant a paralysis of the lower half of the body; this you all understand. But when you have such a form of paralysis and you diagnosticate it as a case of paraplegia, without any further explanations or investigations, you have only made a single step in advance; you have not made a diagnosis. You have ascertained the existence of a symptom only, for paralysis is but a symptom of nervous disease,—never the disease itself; and when you come to the conclusion that an individual has hemiplegia or paraplegia, you have arrived at an opinion no more definite than when you have ascertained that a person has a cough or dyspepsia, fever or dropsy.

I have just stated that paraplegia is a symptom, and you may ask me, A symptom of what? It is symptomatic of a change in the normal condition of the spinal marrow. It is rarely, very rarely, dependent upon a disease of the brain, unless this be associated with a disease of the spinal cord. The affections of the spinal cord eventuating in paraplegia may be divided into two principal varieties, *organic diseases* and *functional disorders*.

By *organic diseases* we mean certain structural lesions of the cord from spinal meningitis, myelitis, softening, sclerosis, hemorrhage, tumors, or *protracted* congestion and anæmia, resulting in integral changes; by *functional disorders* we understand certain morbid manifestations derived from an abnormal working of the nerve-centres of the cord, but without any apparent alteration in structure whatever; and should the opportunity for a post-mortem examination offer itself, the most accurate observations with the microscope would not show any evidence of a histological tissue-change or disintegration. Yet for all that, without the possibility of detecting such a change after death, a paraplegia might have been very marked during the patient's life.

This *fundamental disorder* may depend upon a poisoned condition of the blood, as in smallpox or syphilis; or the abuse of the gratification of sexual desires may produce a paraplegia without the evidence of tissue-change, as paraplegia may depend upon an organic lesion, such as an inflammatory affection, the presence of a tumor, softening of the cord, etc., and in these instances there will be very evident and tangible changes after death; or it may depend upon some functional disturbances, and then no investigation would make the slightest tissue-alteration apparent. I presume you all recollect that in affections of the spinal cord attended by paraplegia the invasion of the paralysis is gradual in character and slow of development,—with the exception of hysterical paraplegia and hemorrhage of the cord,—and all these affections I have fully described. In myelitis, for instance, the invasion is slow, and the paralytic phenomena may sometimes be more apparent on one side of the body than on the other; and accompanying the motor paralysis there is more or less complete anæsthesia or impairment of sensation. Another peculiarity of the paraplegia is that the reflex movements are aggravated.

I suppose you all know what *reflex movements* are. When anybody tickles the sole of your foot and you suddenly withdraw it, you do this involuntarily and without any intervention of the will, the cerebral convolutions having nothing to do with the train of phenomena observed. The withdrawal of the foot in this case is an illustration of reflex movement. The movement depends upon an impulse sent from the ganglionic centres of the cord in consequence of some reflected irritation. In paraplegia this func-

tion is much aggravated. An eminent writer on nervous diseases, Jaccoud, contends that wherever this exalted reflex action exists in combination with the so-called spinal epilepsy (which, by the way, is merely a tetanic contraction, or a rigid stretching out, of the muscles), you may be certain the patient is suffering from some organic disease of the spinal cord, and generally from myelitis. I shall not discuss the innumerable conditions which may give rise to paraplegia in different forms; it is sufficient to state that it is usually a result of some form of irritation, softening, or inflammatory condition of the cord, or of a functional or reflex form of trouble. This suggests the introduction of a very interesting study which will occupy us to-night,—*reflex paraplegia*. The word reflex is derived from the Latin *re*, back, and *flecto*, to turn. By reflex paralysis, then, is meant a paralysis which is not dependent for its origin upon any primary disease of the cord, but is of reflex nature, and caused by some peripheral irritation. This occurs when a disease of the uterus, of the bladder, or of the penis, etc., is turned back or reflected to the spinal centres, producing a paralysis; and it becomes your duty, in cases of reflex paralysis, to ascertain the manner in which it has originated. Many eminent authors, Jaccoud and others, contend that the term reflex paralysis is a very inappropriate name, even though it was given by so great an authority as Brown-Séquard; they hold that the term involves a contradiction, because reflex action and excito-motor action are synonymous expressions. Now, the constant and pathognomonic characteristic of reflex phenomena being “movement,” reflex refers to action and to motion; and hence, paralysis referring to loss of the power of moving, to speak of reflex paralysis is inappropriate. Still, we continue to make use of the name, as it is most generally accepted and one to which everybody is accustomed.

Some maintain that reflex paralysis is induced by an anæmia of the blood-vessels in that particular portion of the cord where the irritation is reflected: hence they call it a reflex spinal ischæmia. Jaccoud is probably nearer right when he says that it is simply a protracted peripheral irritation, resulting in paralysis, in a manner which I shall soon describe.

Dr. Hanfield Jones uses the expression “inhibitory paralysis” as applied to this affection. Now, the fact is that it has been known

for a great number of years, by accurate observers, that sometimes a paraplegia exists during life, and after death not a trace of organic alteration in the component structures of the spinal cord can be found. It has been also ascertained that this paraplegia existed generally in conjunction with certain other diseases, especially those of the genito-urinary organs. Now, though this class of affections embraces those most commonly giving rise to the paralytic phenomena known as reflex paralysis, still, the origin may be entirely different, and may be due to certain emotions, to the condition of pregnancy, to the presence of worms in the intestines, to the irritation consequent upon difficult dentition, or to certain neurolytic causes, as the action of cold, of moisture, or of sudden atmospheric changes, or to other causes seemingly inadequate to produce such results. All of these and a goodly number more may occasion paraplegia, the cause of which is not always very apparent, exciting the anxiety of the patient and the solicitude of the physician. There is something always underlying the paralysis,—an originating cause, a peripheral irritation of the afferent nerves; the cause may be situated in the bladder, the uterus, or elsewhere; and not until you remove the peripheral irritation can you make the paralysis disappear. Hence the important fact that a reflex paraplegia is oftentimes readily removed by ascertaining and curing the causative disease.

You all know that an inflammatory condition of the cord is necessarily hopeless, at least in the vast majority of cases, the disease being progressive from bad to worse, and not showing any disposition to yield to any known remedies. But a reflex paraplegia does not obey the same law: it is amenable to treatment; and if you proceed energetically and ascertain the primary disease and the particular afferent nerves coming from the original seat of disturbance in the peripheral part of the body primarily affected, then, and then only, can you expect to cure by preventing the propagation of the irritation to the spinal centres. You all see that this is a very important subject, and a very interesting one. These doctrines of Brown-Séquard have been confirmed by the observations of many eminent surgeons during the war, especially by Drs. S. W. Mitchell, Moorhouse, and Keen, who found, for instance, that sometimes when a wound was located on one side of the body a paralysis existed in the other; sometimes

a man received a wound in one thigh and the other thigh was paralyzed ; or, again, a wound in the right testicle sometimes produced a paralysis of the right anterior tibial muscle and peroneus longus, etc. Occurrences of this character are explicable upon the theory of reflex phenomena, which affords us their only possible interpretation. Older writers, for instance, have pointed out the connection between the presence of tænia in the intestines and a development of amaurosis. And this, also, is explicable only by the law of reflex action.

What reason do we have for believing that reflex paralytic phenomena are produced in this way ? How do we know that reflex paralysis is distinct and different from organic paralysis ? In the *first* place, we have the rapid cure of the paralytic affection after we have relieved the patient of the primary trouble ; as when we cure a woman of paralysis by restoring a prolapsus uteri. Now, suppose a woman is paraplegic, and we discover that she has a retroverted womb, which we replace in its normal position and support by a pessary, and we find that all at once she recovers from the paraplegia ; let us suppose, now, that after a few weeks, or even a few months, we remove the pessary, and on allowing the womb to resume its abnormal position the paraplegia reappears, perhaps in an aggravated form : would not this be plausible proof of the deleterious influence exerted by the retroversion on the spinal centres ? Such instances have happened. Again, we find that in proportion as the original trouble is aggravated, and the transmission of impressions of irritation to the spinal centres is increased, there is a corresponding aggravation of the paraplegia. No better proof of a reflex origin could possibly be required. The very fact that the changes in the intensity of the paraplegia keep pace *pari passu* with the changes in the phases of the primary disease, is presumptive proof that the paralysis is of reflex origin.

Secondly, we shall see that often when a paralytic condition of reflex origin exists, the form of paralysis will present certain phenomena which are quite characteristic of reflex paraplegia, and are in themselves quite distinct from other forms of paraplegia. You see on the blackboard a table prepared by one of the greatest physiologists of the day,—Brown-Séquard. This table shows you the distinctive features belonging to paraplegia due to urinary

irritation, as distinguished from the peculiar symptoms present in myelitis or organic disease of the cord. If you now read this differential table, you will find that the points of difference between the characteristics of organic and functional diseases of the cord are sufficiently marked, and that the distinctions are not merely hypothetical.

But now we come to a very nice and interesting point, namely, How is this reflex paraplegia brought about? When Brown-Séquard first wrote on this disease, it was after having performed certain experiments that made the phenomena very clear to his mind. By extirpating a kidney, or tying a ligature around its hilus, in an animal where he had an opportunity of making proper examinations, he discovered that the operation produced a contraction of the corresponding vessels of the pia mater of the spinal cord; if only one hilus was ligated, the contraction took place upon the same side of the spinal cord. The ligation, he contended, produced an irritation, which was transmitted to the spinal centres, by which the capillaries were contracted and their calibre diminished, thereby causing an anæmic condition in that particular part of the cord. Brown-Séquard came to the conclusion that in reflex paralysis there is always a reflex anæmia of the cord produced by a reflex contraction of the blood-vessels, in consequence of an irritation in a remote part of the body,—the irritation being propagated back to the cord, resulting in a contraction of some of its blood-vessels. But recent investigators, some of the most eminent French, German, and American authors, do not accept these conclusions, but take exceptions to the views of Brown-Séquard, because his ideas of reflex paralysis are antagonistic to physiological action. They contend that they could very well understand how an irritation from the uterus, the bladder, the rectum, etc., might produce a temporary anæmia, but these irritations are constant in character. In reflex paraplegia, according to this theory, there must be a constant anæmia; and the corresponding irritation of the vaso-motor nerves innervating the blood-vessels, under such circumstances, would also necessarily be continuous. Now, they say, there is no tissue of any kind which can remain permanently irritated or contracted for any great length of time, because this would imply a physiological impossibility. Of course there can always be a condition of irri-

tation of the different fibres; but a period of exhaustion must sooner or later occur, and then there will no longer be a contraction of the irritated muscular fibres of the blood-vessels; but, on the contrary, they will dilate, a paralytic condition of the vasomotor nerve-centres inevitably producing a consequent hyperæmia, and anæmia would then be out of the question.

Besides this, we cannot always accept certain inferences from a few experiments, for are the results of all physiological observations constant, similar, and invariable? Do all observers arrive at the same conclusions as Brown-Séquard? Do we not find many investigators perform the same experiments and still arrive at different results?

These, and many other considerations, undoubtedly help to subvert the theory of a spinal anæmia in reflex paralysis.

Again, Dr. Radcliffe, in J. Russell Reynolds's "System of Medicine," on the subject of reflex paralysis, states as an objection to Brown-Séquard's theory the fact that whilst the symptoms of reflex paraplegia and of paraplegia from myelitis are very distinct, the symptoms of spinal reflex paralysis and those of spinal *congestion* are almost identical. The symptoms of spinal congestion imply a very different pathological state from that of anæmia, and although Brown-Séquard claims anæmia to be the pathological condition of reflex paraplegia, still, it appears that its symptoms are very similar to, and almost identical with, those of spinal congestion; so that we have here two different and antagonistic pathological conditions producing the same symptoms.

S. W. Mitchell, Moorhouse, and Keen *first* advocated the doctrine, claiming that the paralytic phenomena of reflex paraplegia are due to the fact that there is a primary irritation of some peripheral part,—the uterus or any other; that this primary irritation is propagated along the afferent sensitive nerve until finally the impressions of irritation are received again and again; at last, reaching a climax, the spinal nerve-centres become *exhausted*, and as a result of over-stimulation through the sensory filaments of the nerve and the exhaustion of the nervous centres, a paralytic condition ensues, reflex in character, and constituting what is termed reflex paraplegia. Dr. Jaccoud fully concurs with them in their views of the pathology of reflex paraplegia. They cannot receive too much credit for their careful observations and researches.

This opinion not only seems to be rational and easy to understand, but it is also entirely in conformity with the best modern physiological views. As a matter of course, wherever there is undue stimulation there must be a corresponding subsequent depression: wherever there is exaggerated action there must be recoil, reaction, and exhaustion. Some portions of the spinal centres receive impressions of irritation for a certain length of time; they become irritated until they are finally exhausted; they are no longer able to send out a nervous influence of a proper kind, and a paralysis consequently ensues. This is the interpretation of many of the best modern authorities. It now seems quite easy to understand how a simple gonorrhœa may result in reflex paralysis. We can readily conceive that in an individual so predisposed the constantly-recurring irritative impressions from the inflamed urethra are transmitted along the nervous fibres to the spinal centres until these are annoyed, jaded, over-irritated, and exhausted; and as soon as this expenditure of vitality of the nervous centres ensues you will have evidences of paralysis. No matter whether the irritation be due to a gonorrhœa, intestinal worms, troubles of dentition, or uterine or pelvic affections, the mode of action will be the same as soon as the irritation is sufficiently intense to overwhelm the nerve-centres: they will become exhausted and depressed, and will no longer be able to perform their normal duties.

Now you have the whole history of spinal reflex paralysis. I have told you the different manners of manifestation of the disease, and I hope you will recollect that there is a *primary cause of irritation* in all cases. It might be referable to a great many organs, thoracic, abdominal, or pelvic; but it is a fact that some primary disease is invariably present, whether detected or not.

That it is important to cure reflex paralysis is evident from the fact which I told you of in connection with certain functional brain-diseases, and which holds good in spinal diseases, namely: any abnormal condition of the nervous centres purely functional in character, though at first slight and apparently trivial, will, if continued and allowed to exist beyond a certain limit, lead to an impairment of the organic structures, and will in the end produce most disastrous results. In this way it is that a disease entirely functional in character, and of little magnitude in the beginning,

may, by not being properly and timely dealt with, and by being allowed to continue too long, result in organic changes and a subsequent incurable myelitis.

Even a clot in the brain may occasion no inflammatory action, and may become encapsulated; still, if the brain does not become accustomed to its presence, the tolerance ceases, inflammatory changes ensue, and serious organic lesions of a grave character will supervene.

Before giving you the treatment, in summing up I shall say a few words as regards the difference between the most prominent and characteristic symptoms of myelitis and reflex paraplegia.

In reflex paraplegia the paralysis is incomplete: there is no change in the appearance of the limbs, no atrophy; there is no impairment of the muscular contractility, no involvement of the sphincters (unless prior to the paralysis), no tendency to the formation of bed-sores, and no alkalinity of the urine; all these symptoms being characteristic of myelitis. Neither is there any sensory impairment; or, should it exist, it is hardly appreciable. There is little or no anæsthesia, which is characteristic of myelitis, nor hyperæsthesia, which is characteristic of spinal meningitis and of hysteria. Besides this, the paraplegia is incomplete and not decided, while in myelitis, which is a progressive disease, the paraplegia is soon well marked and developed. In fact, the superficiality of the paralytic phenomena in reflex paraplegia, as in hysteria, cannot escape close observation. I have no time to analyze every one of the different features of reflex paraplegia, but I give you the table of *differential characters* by Brown-Séquard. Its perusal will surely repay you, and you will find that it will enable you to make a diagnosis in the vast majority of cases:

PARAPLEGIA

DUE TO URINARY REFLEX IRRITATION.

1. Preceded by an affection of bladder, kidneys, or prostate.
2. Usually lower limbs alone paralyzed.
3. No gradual extension of the paralysis upwards.

DUE TO MYELITIS.

1. Usually no disease of the urinary organs except as a consequence of the paralysis.
2. Usually other parts paralyzed besides the lower limbs.
3. Most frequently a gradual extension of the paralysis upwards.

PARAPLEGIA

DUE TO URINARY REFLEX IRRITATION.

4. The paralysis is usually incomplete, an extreme debility or weakness of the limbs rather than paralysis.
5. Some muscles more paralyzed than others.
6. Reflex power neither much increased nor completely lost.
7. Bladder and rectum rarely paralyzed, or at least only slightly so; sphincter ani weak.
8. Spasms in paralyzed muscles extremely rare.
9. Very rarely pains in the spine, either spontaneously or on application of pressure, percussion, or a hot moist sponge, or ice.
10. No feeling of pain or constriction around the abdomen or chest.
11. No formication, pricking, or disagreeable sensations of cold or heat.
12. Anæsthesia rare, the tactile sensibility being but slightly, if at all, impaired, but the muscular sense is almost lost.
13. Usually obstinate gastric derangement.
14. Variations in the degree of the paralysis corresponding with changes in the primary disease.
15. Usually the urine is acid, unless the urinary organs are diseased.
16. Cure of the paralysis frequently and rapidly obtained, or taking place spontaneously after a notable amelioration or cure of the urinary affection.
17. Usually muscles do not become atrophied, and temperature is little lowered.

DUE TO MYELITIS.

4. Very frequently the paralysis is complete.
5. The degree of paralysis is the same in the various muscles of the lower limbs.
6. Reflex power often lost, or sometimes much increased.
7. Bladder and rectum usually completely paralyzed or nearly so.
8. Always spasms, or at least twitchings.
9. Always some degree of pain existing spontaneously, or caused by external excitations.
10. Usually a feeling as if a cord were tied tightly around the body at the upper limit of the paralysis.
11. Always formications, or pricking, or both, and very often sensations of heat or cold.
12. Anæsthesia very frequent, and always at least numbness.
13. Gastric digestion good, unless the myelitis has extended high up in the cord.
14. Ameliorations very rare, and not following changes in the condition of the urinary organs.
15. Urine almost always alkaline.
16. Frequently a slow and gradual progress towards a fatal issue, and rarely a complete cure.
17. Atrophy of the muscles of the paralyzed parts.

The next question to consider is, How are we to treat cases of reflex paraplegia? What is the most appropriate method of treatment to relieve the disease? I presume the answer will suggest itself to all of you. If any original or primary disease or discoverable difficulty leads to the production of the paralytic phenomena, common sense and sound judgment will demand its removal. Therefore, if a paraplegic woman has an inflammation of the mucous membranes of the urethra, the uterus, or the vagina, you will essay its relief by appropriate treatment, especially by the use of anodynes locally; or if the original trouble is caused by the presence of worms in the intestines, try to expel them by anthelmintics, etc. Now, though the eradication of the primary disease often cures the reflex paraplegia, still, the trouble is not always over, especially if the paraplegia has lasted for some time; for, the nervous centres being exhausted, they necessarily require toning up. From what I have told you of reflex paralysis, you of course know that the spinal centres are depressed: it is not simply from an anæmic or hyperæmic-state of the cord, but from an exhaustion produced by prolonged irritation, and you readily see that the internal (not the local) use of ergot, belladonna, or bromide of potassium must, for obvious reasons, have disastrous consequences: these remedies cannot fail to do harm, for they tend to diminish the quantity of blood in the cord; and if to an exhaustion of the nervous centres you superadd an anæmia, you intensify the trouble, only adding fuel to the flame. But if you wish to remedy this exhaustion, have recourse to strychnia, the remedy *par excellence*, to the chalybeate and arsenical preparations, to quinine, and to electrical stimulation by mild forms of either the continuous or the faradic current, which in addition are serviceable in stimulating the muscles so long inactive. In conclusion, I shall repeat once more the point: bear in mind that whenever you have a paraplegia due to a reflex transmission of irritation to the spinal centres, you can never hope to cure the paralysis unless you relieve the patient of the primary and causative affection.

LECTURE XXXII.

CONVULSIONS.

●
Causation.—Divisions.—Sequelæ.—Morbid Anatomy.—Convulsions in Children.—
Premonitory Symptoms.—Treatment.—Apomorphia.—Calomel.—Santonin.—
Sudden Interference with Cerebral Circulation.—Remedies.—Convulsions in Adults.
—Epilepsy.—Uræmia.—Cerebral Hemorrhage.—Injuries to the Head.—Cerebral
Aneurism.—Intra-cranial Diseases.

GENTLEMEN,—I have selected for to-night's lecture a subject replete with interest and importance,—convulsions. By convulsions we mean an alternate succession of tonic and clonic spasms, in a manner which I shall fully describe. As I have already, in a former lecture, given you the difference between these two forms of spasmodic contraction, it would be useless to encroach upon your valuable time by the reiteration of facts already well known to you. All that I shall say in this connection is that the best example of a tonic spasm is found in tetanus, while clonic spasms are best shown in hysteria. We can therefore consider these two affections as typical of the two different morbid phenomena which we are now studying. As a general rule, all convulsions are more or less tonic at first, and we have the clonic spasms remaining or alternating with the former.

The first important fact I have to communicate in regard to convulsions is, that when they occur they are never the direct evidence of anything but the existence of a *symptom*. Therefore, when you have ascertained the presence of convulsions, you have established nothing more than when you have learned that a patient has dropsy, fever, cough, or paralysis; that is, you realize the existence of a certain symptom, and nothing more. This point I wish to impress upon your minds; for the symptom known as convulsion is common to several pathological conditions or to many different morbid states, and consequently subject to many different interpretations; and this is important to understand in view of the treatment, as we shall hereafter see.

Let us inquire into the ordinary *causes* of convulsions. In the first place, any organic disease of the brain (and here we have a very wide field), any organic cerebral disease, whether an inflammation, or a softening, or an adventitious growth, or an intracranial affection of any kind, may cause convulsions. Then certain traumatic causes, such as blows, falls, or other injuries to the head, may have the same effect. Again, both an anæmic and a hyperæmic condition may be accompanied by convulsions. So it is that women dying from a severe loss of blood, as in post-partum hemorrhage, generally have convulsions before death; while, on the other hand, persons suffering from an intense congestion of the brain are equally liable to them. You see that certain diametrically opposite pathological conditions of the brain may lead to the same convulsive phenomena. Then, sometimes in pregnancy we have eclamptic attacks, due to a morbid influence exercised through the blood on the nervous centres, occasioned by an improper performance of the functions of certain organs of excretion and secretion. Again, several kidney-diseases, especially the affection in which we have the small, contracted kidney, may lead to the development of convulsions by the non-elimination of urea from the blood, producing the pathological condition known as uræmia, or uræmic poisoning. Besides these, we have many causes of different origin, such as the exanthematous diseases, violent emotions, the presence of worms in the intestines, or an accumulation of ingesta in the alimentary canal: these, and also a difficult or irregular dentition, may give rise to convulsions. The same result may be produced by any of the cachexiæ, especially the syphilitic; also by certain poisoned states of the blood, whether from mineral, animal, or vegetable poisons, or diathetic conditions. All these may lead to the development of this common symptom. Again, if we are unable to classify the convulsions, if we are ignorant in regard to their nature, we call them *idiopathic*, *essential*, *epileptic*, or *eclamptic*. The nerve-tissues invariably suffer during a fit, there being always an excessive expenditure of nervous force in the nerve-cells; and the spasms or convulsions are always accompanied by more or less nutritive tissue-change, a greater or less structural retrograde alteration, temporary, but depressing and exhaustive in character. When we carefully and critically analyze and investigate the phenomena

of convulsions, we ascertain that they are simply the outward signs or manifestations of many different morbid conditions. They are only the indications or outcroppings of some violent inward commotion, of some intense internal trouble or perturbation in the economy; these manifestations being alike under varied circumstances, regardless of the dissimilarity of the different causes producing the convulsions. Hence the meaning of a convulsive attack, or the deductions to be drawn therefrom, are not always so clear and plain as you might have supposed. It is by no means easy to know what to do when they are present; and we would surely not be at a loss were little to be sought for beyond the convulsive phenomena themselves. Still, you may believe me when I say that their origin and cause are often much more difficult to ascertain than the etiology of a dropsy or a dyspnoea.

Before entering any farther upon the discussion of our subject there is one thing which I will do: it is to follow Dr. Hughlings Jackson's division of convulsions into *two grand distinctive groups*. This will facilitate our study and answer very well for practical purposes. We classify them as occurring in young children, and in persons from the age of seven upwards. Now, convulsions, whether in children or in adults, are often evidence of the same pathological condition of the brain or spinal cord; but the physiological meaning is vastly different, and has a very wide range of interpretation. It is not very difficult to realize this, if you observe that the phenomena of convulsions in children are much more limited and partial in character than in adults. Take, for instance, the carpo-pedal contractions or spasms which occur in infants, and you will see that the limitation of the spasm to certain particular parts is well marked and defined. You have the characteristic distortions of the wrist and ankle, the bending inwards of the thumb, the strong contraction of the toes, etc. But why is it that we have this greater limitation of the convulsive phenomena in children than in adults? This is, in all probability, due to the fact that in children the particular parts of the nervous centres which preside over the innervation of the muscles are not so well developed as in adults; and again, as Hughlings Jackson says (from whom I borrow many prominent ideas upon this subject), "there is probably a more specific attraction in particular localities for certain morbid processes in

children than in adults." The reason of this is that the cerebro-spinal centres, composed of both the cerebral and the spinal centres, "are not knit together so thoroughly" in children as in adults; and, consequently, there is always a tendency to the limitation of the pathological processes to some particular portion or group of nerve-centres. Then, also, the great difference of physiological interpretations of convulsions in children and in adults is due to the preternatural mobility and instability of the entire nervous system in the former. There is, according to West and to Trousseau, an undoubted predominance of the spinal over the cerebral system in childhood; therefore, in most of their pathological conditions, the ordinary phenomenon is—what? Delirium? No; it is not. Little children have hardly any mind that can become perturbed in action, having as yet too little power of imagination to allow the mind to wander in the manner called delirium. Their different movements do not require the emanation of volition from the ideational centres, but are nearly entirely *spinal* in origin, and therefore involuntary, automatic, and reflex in character. This accounts for the greater liability of children to convulsions, which are nothing else than muscular movements not checked or controlled by the powers of the will. When the newly-born babe feels the instinctive cravings of hunger, it seeks for the nipple of the mother's breast merely by an automatic act, depending for its stimulus upon the spinal, not the cerebral, centres,—the automatic action being under the control of certain centres situated deeply in the substance of the spinal cord. Now, when the child is sick, this spinal control and equilibrium are disordered, and we have the alternate contraction and relaxation of the voluntary muscles, known as convulsions. Therefore it is that when an adult, under the depressing influence of the malarial poison, has a fever preceded by a chill, the chill will be replaced by the corresponding phenomena of convulsions in an infant. Again, the pathological conditions giving rise to delirium in the adult lead to convulsions in young children; and, if we follow Trousseau, we shall see that even dreams in adults may be supplanted by convulsions in children; in other words, what would be dreams in children are sometimes manifested, instead, by convulsions.

In regard to the advent of convulsions at different periods of life, you may set it down as a rule that in proportion as the indi-

vidual advances in age the tendency and liability to convulsions grow less; and, also, in proportion to the youth of the person will the development of the convulsions be more partial and limited in character. Hence it is that in children we often have a spasmodic contraction of a single muscle, as the biceps, or a group of muscles, as in the carpo-pedal contractions, or in the affection of infants called *false croup*, where we have a convulsion of the diaphragmatic muscles and auxiliary muscles of respiration, such as the pectorals and intercostals; then, also, the laryngeal muscles, so that we have *laryngismus stridulus*, an affection consisting simply of convulsive phenomena in consequence of the perturbation of the nervous centres presiding over the different muscles I have just enumerated, and accompanied by a peculiar crowing sound, which takes place at each deep inspiration. The tetanic rigidity in which we sometimes find those muscles of respiration is always a source of great danger to the child, of intense anxiety to the mother, and of deep solicitude to the physician; for, if not relieved, it invariably has a tendency to produce death by asphyxia. But the stridulous sound itself is always a source of joy to both mother and physician, as it is the best evidence of the breaking up of the tetanic spasm of the laryngeal muscles, showing that the child is able to make the necessary inspirations, and that the danger is over, at least for the present. These paroxysms may take place once a day, or even ten, fifteen, or twenty times a day, and of course there is always great danger of the child dying in one of them. Some time ago I had under my care a severe case of this nature. I first directed my attention to the general condition of the nervous system; and, after having sought to correct any of its irregularities which were obvious as causes of the attack, I cured the child by the administration of hydrate of chloral in combination with bromide of potassium. These, if carefully given, are invaluable and reliable remedies.

Now, suppose you are called upon to treat a child or an adult in convulsions. The first thing to do is to study the paroxysm; the second, to take into consideration all the circumstances explanatory thereof, such as the different conditions surrounding the patient, or anything that may give you a clue to the correct interpretation of the convulsive manifestations. You may observe, for instance, that the convulsion is partial or limited in character, or

there may be a general convulsive condition of the voluntary muscles, contractions both tonic and clonic. Then you study the violence of the attack and the tendency to a repetition ; or, better, the intervals between the paroxysms. You take into consideration every fact or link furnished by the convulsive paroxysm proper, and then you pass to the study of the particular features of the case which may possibly throw light upon the subject. Of course the convulsion itself does not furnish any light, for, like fever, cough, etc., it is only a symptom ; but the study of the paroxysm is always important, because any abnormal muscular action, whether slight or violent, is always indicative of a nervous trouble of some kind in the background. The contractions may be due to a grave and irremediable cerebral lesion, or they may be the consequence of only a slight nervous irritation or disturbance ; the cause may be so trifling as hardly to be realized : a simple flatulence in the intestines, or an accumulation of ingesta, may lead to muscular twitchings, and, in persons whose nervous elements are in an unstable condition, even to general convulsions.

The next thing to be considered is the *sequelæ* of convulsions in adults and in children, and these are always difficult to explain. In some children an amaurosis develops after convulsions ; in other persons there is a deficiency of memory, or of speech, or a subsequent discontinuance of mental development. Again, children subject to convulsions are apt to squint, or there may be some other paralytic phenomena, such as hemiplegia. Now, whether hemiplegia is the result of convulsions, or whether the convulsions are the result of the same pathological cerebral lesion that produces the hemiplegia, are questions yet obscure, and we are not able to solve them. It is not, therefore, necessary to discuss them ; but as practical physicians you should know that certain eclamptic attacks are sometimes followed by hemiplegia and other complications. A peculiar fact in regard to convulsions is the morbid anatomy, for, notwithstanding that a child may have died in an eclamptic attack, we may, upon post-mortem inspection, find no evidence of any structural change in the cerebro-spinal or other organs of the body. Now, I do not doubt that a certain change may exist ; but it is not made apparent by our present means of investigation. We can easily understand and appreciate

this fact if we consider that convulsive phenomena may result in death when the little patient is asphyxiated or the spasm is prolonged beyond his endurance. A single spasm may thus kill a child, while he may survive many spasms of less intensity. The fact of the morbid anatomy being so obscure shows our great ignorance of the subject of convulsions. By the study of the paroxysm we have derived but little information regarding the diagnosis or prognosis; but let us examine the clinical history and the different circumstances surrounding the patient in a given case of convulsions. Suppose you are called upon to see a person thus affected. Of course the whole household is upset, and everybody is laboring under intense excitement and fright. You will see, in the first place, certain clinical phenomena, which as practical physicians you will at once seek to unravel. You will say to yourselves, Can I get any clue to understand this or that symptom by the study of any centric or eccentric cause? You question the relatives as to whether the little patient has been eating anything to which its stomach was not accustomed, or whether it has been overloaded with food, whether it has been constipated, or whether they have noticed that the child has been passing worms; or there may have been some troubles of dentition, and you may examine the gums yourselves. Still, I believe, with Dr. Jackson, of London, that fright is a not uncommon source of convulsions in children, and one which is very often overlooked. When the trouble arises from worms, constipation, dentition, etc., the convulsions do not recur after the cause has been removed; but if produced by fright or other emotion, there is a great liability to recurrence in subsequent years from any nervous trouble. Convulsions are a symptom, and may be a symptom common to many different diseases. You all know, from what I have said of the great instability and unusual mobility of the nervous centres in children, that the period of invasion of any acute affection, whether inflammatory or otherwise, may be marked by convulsions. You feel the skin, which may be hot and dry; you measure the temperature by the thermometer, and find that it is above the normal standard. You think of meningitis. You know that convulsions are often precursory of a coming storm, such as meningitis, scarlatina, pneumonia, or typhoid fever. Again seeking all possible information, divest the child of its clothing, auscultate the chest,

and endeavor to elucidate all doubts suggested to your mind. In eruptive diseases, the abnormal temperature of the body and skin, the frequent, rapid pulse, etc., are significant, being especially the precursors of a scarlatinous rash which will soon follow. But a fit may have some connection with a perverted condition of the child's health, and the mother may inform you that the poor little sufferer has been subject to some exhaustive form of disease, some protracted and depressing discharge, such as diarrhœa, or some continued gastric derangement, with vomiting. There may be a general anæmia, and an anæmic condition of the nervous centres; in fact, the child may suffer from hydrocephaloid disease. Now, considering that the convulsive phenomena in this disease are precisely the same as in congestion of the brain, you see how disastrous the consequences may be if the physician mistakes, and treats the hydrocephaloid disease for cerebral hyperæmia. But what is this hydrocephaloid disease? It is a pathological condition described by Marshall Hall, and of which I have fully spoken in a former lecture. I told you it was caused by an anæmia the result of a continued diarrhœa, cholera infantum, or protracted intestinal trouble of some kind. The nervous centres being no longer nourished, their vitality is lowered, and hydrocephaloid disease and convulsions are the natural consequences. This anæmic condition is of therapeutic importance, because stimulation by good nutritious and easily-digested food is required, as opposed to depletory measures, which are resorted to by some in hyperæmic conditions of the brain. Then the convulsions may be symptomatic of some chronic brain-disease,—the result of intra-cranial tumors, abscesses, or softening, or of some recent or old meningeal trouble, etc. The most violent convulsions I ever witnessed initiated a severe pneumonia in a little girl.

You have now seen the many different conditions which may lead to convulsions in children; and when called upon to act in an emergency, you can understand the many questions which must present themselves, even to the most experienced, learned, and eminent physicians, in regard to the causation and treatment. Again, the convulsions may be epileptic in nature, or derived from a previous epileptic condition: in this case you cannot reconcile the phenomena with any other cause, the history of the case pointing unmistakably to the epileptic character of the symptoms.

In the study of convulsions there are always three particular points to consider. I shall not dwell very long upon them, for I have but little time and must condense. The first point is, always, the age of the patient; the second, the previous condition of health,—whether the convulsions occurred while the patient was in apparent health, or was he suffering from some acute malady, or from chronic and wasting discharges, as in hydrocephaloid disease? The third point is, the evidence presented by the peculiarities of invasion of the convulsive phenomena,—whether it was slow and gradual, or sudden and violent; whether the convulsions appeared, ceased, and never returned, or followed one another repeatedly, even without intermission, as in the *status epilepticus*. In the latter case children soon become comatose and fall into their last profound sleep. Now, the points just enumerated you must always take into consideration: *first*, the age of the patient; *second*, the previous condition of health; and *last*, though not least, the mode of invasion.

There may be certain *premonitory symptoms*, or there may not; and when they exist they vary considerably in character. There may be internal twitchings, certain contractions and distortions of the facial muscles while the child is asleep; or there may have been insomnia for some time, and this is a symptom considered by Dr. Churchill of great importance. Then, again, a severe eclamptic attack may occur without any precursory symptoms, so that though the prior occurrences may be important they do not always furnish much information. Avail yourself of everything that may help to clear up the mystery; strip the child and auscultate the chest, so that you may derive all the information which experience may furnish, and so as not to be obliged to remain inactive, but to be able to have recourse to judicious and appropriate measures. Never forget the fact that fright is a common cause of convulsions in children. This is often caused by foolish servants or indiscreet people working upon the imagination of little ones with terrible stories; their emotional powers become so abnormally sensitive that often after they have gone to sleep a slight noise throws them into a state of terror, with convulsions following if the least instability of the nervous system exists.

The connection of convulsions with epilepsy is a very important matter. You will more fully realize this in your practice, for one

of the first questions asked of you when visiting a child subject to convulsions is, "Doctor, is this epilepsy? Will the little one in after-life be subject to a convulsive form of disease?" It may be generally stated that when convulsions occur at distinct intervals, good health being enjoyed between the attacks, they are a source of grave solicitude and anxiety to the physician, especially when he is aware that a certain predisposition to nervous disease exists either among the relatives or ancestors; it is almost conclusive proof that the convulsions are epileptic in character. Now, convulsions that are not epileptic do not often become so in children; but, on the other hand, when they repeatedly suffer from them, they work injuriously upon their nervous system, which necessarily is already more or less at fault; consequently, when advancing in age and subjected to the vicissitudes of life and the incessant struggle for self-maintenance, they are very liable to suffer from the development of some disease of the nervous system. Of course, to give a clear prognosis in such cases and to predict the future condition of health of such a child would be an utter impossibility, and it is therefore better to give a qualified opinion.

This brings us to a most interesting point, the *treatment* of infantile convulsions. I have not the time or the inclination to enumerate all the views held by the different authors upon the subject, and shall simply give you my own opinion. From what I have told you, you know that when a child has an eclamptic attack you should always examine its gums. Should you discover any trouble of dentition, they should be immediately lanced, and in this manner you may afford the necessary relief. Again, if you should have a case like one I remember having had in my practice a few months ago, and the mother should tell you that the child was stuffed with hickory-nuts, molasses-candy, and dough-nuts, you should try the administration of an emetic. Now, when a child has an eclamptic attack, it is not always an easy matter to produce an evacuation of the stomach, for it may be in a state of unconsciousness and unable to swallow; but do so as soon as possible; or, should the ingesta have passed into the bowel, give an enema, together with other remedies which I shall mention in an instant. When giving an emetic, always be careful to give one that will act rapidly and produce as little prostration as possible. Apomorphia, hypodermically injected, acts energetically and

efficiently, and should be essayed when the child is unable to swallow. If the mother tells you that she has observed the presence of worms in the passages from the child's bowels, the probability is that they are the cause of the convulsions, and you should give the usual anthelmintics; and in this case calomel is to be selected, for it is easy of administration, and the child can swallow it without any effort, neither does it throw it up; and the remedy seems to have a marked influence in causing the death of the worms, and their expulsion: it should usually be combined, under such circumstances, with santonin. In all cases of cerebral irritation from a hyperæmia or inflammatory disease, you can have recourse to purgatives and cold affusions to the head. Cold affusions are especially recommended, by high French and German authorities, when the convulsions are symptoms of an impending scarlet fever. Under these circumstances convulsions are symptoms of irritation and not of depression, and are accompanied by an elevation of temperature dangerous to life. Now, though I am decidedly opposed to the so-called antiphlogistic treatment of convulsions, yet, where convulsive phenomena are symptoms of irritation accompanying certain brain-diseases, and where the brain is still in an irritated condition, I believe that cold affusions will do good; but in general anæmia or hydrocephaloid disease they cannot fail to do harm. Hence you see the importance of obtaining a thorough history of the case before proceeding to the treatment.

Another remedy frequently used is the warm bath: for this purpose the water should be heated to a maximum of about 100° F. If some eruptive disease is lurking behind the convulsions, the warm bath will often have a soothing, calming, quieting, and sedative effect; and if in combination with the muscular spasms there are symptoms of cerebral origin, pointing to congestion of the brain, and you have both an incipient exanthematous disease and a cerebral hyperæmia to deal with, you may safely use the warm bath, and also cold affusions to the head whilst the child is immersed in the bath. If you have any decided evidence of a mechanical congestion or hyperæmia of the brain, a leech or two, in rare and exceptional cases, applied to the mastoid process of the temporal bone, may be beneficial. Never forget that although you may have convulsions accompanying a hyperæmia of the

brain, still, the pathology of convulsions is such that in most cases where children perish therefrom no trace of a structural change can be found on post-mortem examination. Now, considering that the symptom of convulsions may be the result of antagonistic morbid conditions, of an anæmia as well as of a hyperæmia, of a toxæmia or of a *sudden interference with the circulation in the brain* (and this, by the way, is not an uncommon cause), it is evident that the use of spoliative remedies in all cases must work harm; for though in one case it might perhaps do good, it would inevitably be death in another. In hydrocephaloid disease, do not take the poor baby and dash cold water over its head, do not stuff it with calomel or emetics, do not use any harsh remedies, but handle it carefully and gently, give it a slight stimulant, a little brandy diluted, and put it to the mother's breast. If it refuse to take it, give it some good and nutritive juices, try to keep up the vital spark which is so nearly extinguished, and by this mode of treatment, by tender nursing and removing the cause of the original disease, you may prevent a disastrous result.

Well, you probably will ask me, have we no *specific remedies* by which to subdue convulsions?—some remedy that we might invariably rely on in all cases? In answer, I will say that we have innumerable and vaunted so-called specifics, so many that it would take too long to enumerate them; but I shall simply tell you that I have faith in only one,—the *bromide of potassium*. That this has a remarkably quieting effect upon the nervous system I firmly believe, for experience has convinced me of its efficacy; *but it is not applicable to all cases*; and we are, moreover, ignorant of the particular manner in which it sometimes acts. That it produces an anæmia in the nervous centres all will admit; that it diminishes an irritation of the spinal system, especially its *reflex excitability*, there is no doubt. If you cannot give it by the mouth, inject it into the rectum, and you will often find that it has an equally beneficial effect. Hyoscyamus and assafetida are highly recommended, and you can try them if you will; but I prefer the bromide of potassium.

I shall now, following Dr. Hughlings Jackson's order that I have adopted in this lecture, rapidly refer to *convulsions in persons over seven years of age*.

The first condition which may lead to convulsions is *epilepsy*. I have already said so much about epilepsy that I believe you are fully conversant with its different phenomena. You will determine its presence by the history of the case, and by the patient's biting his tongue, sometimes passing his urine involuntarily, and falling into comatose conditions for a short time after the fit has passed away.

Then convulsions may be caused by *uræmic poisoning*; and in such cases what do you expect to find? You will first have the history of kidney-disease; but if there is no evidence of this, either from the observation of the patient or information from his relatives, you may easily settle the question by putting a catheter into his bladder and examining the urine microscopically for casts and blood-disks, whilst at the same time you test it chemically for albumen by heat and nitric acid: if there is a coagulation your suspicions of convulsions from uræmia are confirmed. Therefore, always beware of Bright's disease, for in this affection the kidneys do not properly perform their excretory functions: they do not eliminate the urea from the blood, and the latter, poisoned by the presence of this excrementitious substance, acts upon the cerebral centres, not only producing convulsions, but also ending in coma, with a fatal result if not soon relieved. Hence the convulsions of uræmia are only the precursors of fatal coma. But the history of the case is generally sufficient to establish a diagnosis.

We may sometimes have convulsions the result of *cerebral hemorrhage*. In this case we also may have convulsions before the coma. Of this pathological condition I have said enough in a former lecture to preclude the necessity of any lengthy description here. You have the age of the patient, the degenerative changes in the tissues, the *arcus senilis*, the peculiar condition of the peripheral arteries which I have so often alluded to, and the convulsions, followed by marked and complete hemiplegia, and accompanied by profound coma, in which there is a remarkable guttural sound during respiration. All these are so characteristic as to enable us to recognize the disease whenever convulsions exist, which in cerebral hemorrhage may or may not be the case. In uræmic coma there is also a noisy breathing, but the notes are not so high in pitch as in cerebral hemorrhage; and in neither case do we find the bitten tongue, as occurs in most cases of epilepsy.

A *severe blow or injury to the head* may lead to convulsions in adults. This may happen in the street. You may be called upon to see a person in a state of unconsciousness, and the bystanders may only be able to tell you that the **individual had a fit** when they first saw him fall; that is all they know about the case. But here, again, though it may at first be difficult to determine whether this is a case of epilepsy, or of cerebral disease, certain external evidences, such as blood oozing from the nose and ears, and, in the case of injuries of the vertex (which are not so common in epilepsy), will point to the fact that the fit was caused by a blow, or of some injury to the head. A post-mortem examination, and upon opening the calvarium, will afford evidences of rupture of a cerebral aneurism, or of some other cause of the last fit of his life. Now, understanding this, it is not so difficult to fall in a fit on this floor before your eyes, as it is in a fit of epilepsy, it might be an impossibility to state the cause of this fatal occurrence, if nothing whatever was known of the previous history or of the different characteristics of the fit.

An adult may also have a fit the result of *cranial disease*; he may have tumors, or other diseases of the brain. However, the other symptoms of intracranial disease, such as headache, vomiting, &c., will explain, the different peculiar characteristics of the fit, so evident, as necessarily to lead to a cor-

LECTURE XXXIII.

CEREBRAL TUMORS.

Natural Division.—Vascular Tumors.—Parasitic Tumors.—Constitutional Tumors.—Accidental Tumors.—Appearance of Symptoms.—Irritation: Direct and Reflex.—Intermission of Symptoms.—Three Forms of Symptomatic Indications.—Fleeting and Persistent Symptoms.—Ordinary Symptoms.—Cephalalgia.—Psychical Symptoms.—Cerebral Apathy.—Vertigo.—Bizarre Sensations.—Symptoms of Mesencephalic Excitation.—Vomiting.—Constipation.—Circumscribed Local Symptoms.—Febrile Reaction.—Transitory Paralysis.—Peculiarities of Paralysis.—Impairment of Reflex and Electrical Contractility.—Topographical Bearings of Lesions.—Impairment of Nerves of Special Sense.—Impairment of Intellect.—Recapitulation of Symptoms.—Diagnosis.—Prognosis.—Treatment.

GENTLEMEN,—We have now to consider the subject of *cerebral tumors*. It is a theme replete with interest, but it is also an intricate and difficult one. I shall endeavor to divest it of much of its obscurity, and make it as clear and intelligible as possible. Cerebral tumors, in the first place, are always limited but persistent pathological lesions,—limited as regards the space occupied, persistent as regards their duration. The existence of a tumor in the brain will, of course, have two different results,—those of direct and those of indirect pressure; the former being produced by the actual contact, the immediate pressure of the tumor upon the cerebral pulp, while the indirect pressure is secondary, and due to the propagation of the direct pressure to other parts of the brain. Strange as it may appear, the *volume* and the *seat* of the tumor are far more important in pathological significance than is its mere *nature*. You will easily understand that any foreign body or growth situated in the neighborhood of the crura cerebri, the pons varolii, or the medulla oblongata must necessarily be followed by more disastrous and injurious results than if it were located in the medullary masses: hence the seat of the tumor is of great importance. That the volume of a tumor, and the rapidity with which it increases, are also of importance is self-evident, and requires no further demonstration.

Having established the fact that it is not so necessary to ascer-

tain the nature of a tumor as to determine its seat, its volume, and the rapidity of its growth, we are now brought to the *natural division* of cerebral tumors, first premising that only exceptionally do they arise from the meninges.

It is not my desire to burden your memory with an elaborate description of the intimate nature and pathological minutiae of different tumors, or of the many varying pathological and anatomical characters which they may display, for this would only be a source of perplexity to you, and, being soon forgotten, would not help you in the least in the practical consideration of the subject. The division I have selected is from Jaccoud, from whose article upon the subject I have extensively borrowed: it has the merit of being simple and easily retained. He divides cerebral tumors into *vascular, parasitic, constitutional or diathetic, and accidental tumors*.

As regards the *vascular tumors*, we divide them into ordinary erectile tumors, and aneurismal dilatations of the cerebral arteries. The vessels most likely to be affected are the basilar artery, and several others at the base of the brain,—not minute arteries, but generally vessels of considerable size and importance. An aneurismal tumor in the brain may be of any of the varieties of aneurisms situated in other parts of the body; it may be simple, mixed, or dissecting. The liability to the formation of these pathological processes is greatest in persons somewhat advanced in years, most generally between the ages of forty and sixty. They are usually the result of some atheromatous change or degeneration in the coats of the arteries themselves, following *endarteritis deformans*, of which I have so often spoken in former lectures. These aneurisms may be of the size of an almond, or larger; and the pressure which they exert on the proximate nervous substances is, in consequence, necessarily disastrous. It not infrequently happens that the aneurism bursts: as a matter of course, this is immediately fatal, and death will be preceded by convulsions,—a fact to which I alluded in my last lecture.

The *parasitic tumors* are divided into ordinary, cysticerci, and echinococci. It is not necessary for me to dilate upon these, since a reference to your books will furnish you with all the desired information.

The *constitutional tumors* we divide into the *cancerous, tuberculous, and syphilitic varieties*.

Among these the *cancerous* variety offers quite a peculiarity, inasmuch as a cerebral cancerous tumor is almost never secondary, but nearly invariably primary, in character. In most cases, therefore, of intra-cranial cancerous growths, the brain is affected before any other part of the body, and the development of the malign tumor is often so rapid that it results in disastrous and fatal consequences before the cancerous cachexia has made its appearance. Should you then rely upon the occurrence of this cachexia for the diagnosis of a cancerous cerebral tumor, you may wait in vain, as the patient will generally be killed by the tumor before the cachexia is developed. Again, as regards cancerous tumors, remember that the fungus hæmatodes, or the encephaloid variety, is the form of carcinoma most commonly met with in the brain.

Should the diathetic tumor be *tuberculous* in character (it usually occurs thus in children), the deposit will generally be in groups or chains; the tubercles are rarely separated, but occur in flocks or clusters, agminated or aggregated, and are of the size of a millet-seed, or larger; they are also found oftener at the base of the brain, or on its convexity, than in its central portions; are secondary, and generally follow tuberculization of other organs, especially the lungs.

Syphilitic tumors are a result of the syphilitic diathesis; they appear sometimes as small nodules, of no very definite shape, but in most cases are merely shapeless, diffused masses of syphilitic, gummy exudation.

The *accidental tumors* consist of different kinds of neoplasms, principally sarcoma, or some growth of fibro-plastic material. These need not occupy our attention, as it is not our province to discuss their nature or peculiarities.

In all cases of cerebral tumors you will necessarily expect and may predict the consequences of a reduction of the intra-cranial space, from the increasing encroachment of the new growth, and also the corresponding manifestation of symptoms more or less grave in character. Tumors, then, besides the simple condition of encroachment, have also certain *reflex* and characteristic results, namely, atrophy, congestion, and reflected irritation. That we may have these different effects is easily explained. You all understand that by the continued pressure of the tumor we must have an atrophy, with disintegration and retrograde molecular

changes, in the histological nervous elements, and their subsequent absorption; but then the brain-mass situated in apposition or in close proximity to the tumor may become irritated, which irritation may produce a congestion. Then, again, we may have a secondary œdema, and an anæmia, and, of course, more or less disturbance of the cerebral circulation, all being the result of the primary irritation caused by the pressure of the tumor; but the irritation from such disturbances may be reflected to distant parts. We have first an atrophy, and secondly a congestion, or, as the French term it, a *turgescence*, following the irritation.

As regards the *symptoms* of cerebral tumor, this is also a very interesting topic. In the first place, a tumor may exist in the brain, and occupy a large space even, yet occasion no particular symptoms during life. Trousseau (the great author whose writings are not alone invaluable so far as nervous diseases are concerned, but are classical and reliable in every department treated upon) relates a very interesting case, cited by Velpeau, of a barber in Paris, in whose brain enormously large cancerous masses had developed themselves: they were situated, I believe, in portions of both anterior lobes. This man was remarkably bright and loquacious, and evinced no symptoms that would give rise even to the bare suspicion of a cerebral tumor. Hence a patient may perish from such a pathological condition without the manifestation of any brain-symptoms whatsoever till during the closing days of life. Heretofore latent, they then become very marked, active, and evident. In others, however, the characteristic symptoms of tumors exist from the very commencement of their intrusion. Tumors will produce symptoms in proportion as the region of the brain in which they are developed is tolerant or intolerant of their presence. As I have already told you, a tumor may exist in the medullary masses, and even attain a large size, without any symptomatic evidence. This happens when the part of the brain involved tolerates the existence of the foreign body, when it does not become irritated by its presence. But if the tumor be developed in the mesocephalon, that is, in the intra-cranial prolongation of the spinal cord, as the medulla oblongata, the pons varolii, the tubercula quadrigemina, and other important ganglia at the base of the brain, or in those parts which in a former lecture I described as the system of conjunction,

namely, the corpora striata and thalami optici,—then, as a matter of course, grave and characteristic symptoms will inevitably result.

Again, cerebral tumors may act in different ways. *First*, when a tumor is pressing on some portion of the brain we may have symptoms which will at once awaken a suspicion of the existence of the encroaching growth. These are symptoms of direct irritation. *Secondly*, we may have symptoms the result of *indirect* or *reflex* irritation. I shall try to make this clear by means of illustration. Let us suppose a tumor in the neighborhood of the origin of the third pair of nerves, immediately implicating the origin of these nerves: as a *direct* result of the irritation caused by the contact of the tumor we shall have certain paralytic phenomena, which always ensue when the physiological functions of a nerve are interfered with. And in this case there will be strabismus, dilatation of the pupil, ptosis, etc. But, independently of this, the original irritation may be *reflected* anywhere else in the brain. It may be transmitted to the convolutions of the hemispheres and give rise to more or less intense psychical disturbances, or the irritation may be reflected to the medulla oblongata and cause an impairment of its physiological functions, or it may be turned back to any other important cerebral organ.

Another thing we have to consider is the facts suggested by the *intermission of symptoms*. The symptoms are not always constant, pathognomonic, univocal, or uniform in character. On the contrary, they are, generally, continually varying, present one day and absent another, and at each time differing in intensity. They are intermittent; and this is a matter of importance to understand. You will easily comprehend it if you bear in mind that we are dealing with symptoms of irritation. In order to elucidate this I shall commence by giving you an invariable physiological law,—namely, whenever there is an undue nervous stimulation or irritation there must arrive, sooner or later, a period of nervous exhaustion; and in proportion to the violence of the irritation will be the intensity of the depression. This depression gives the nerve-power time to recuperate, and whenever its vigor is re-established it will once more lead to the symptoms of irritation, the temporary disappearance of which was due to the exhaustion following the primary irritation producing the interruption or intermission of symptoms, which you now fully understand. It is

not my aim to enumerate every little symptom or feature connected with cerebral tumors. What I desire is to give you the philosophy of the subject; and this you will never forget. Another explanation of this intermission of symptoms is one which we derive from our knowledge of the laws concerning hyperæmia of the brain, on which I have said so much that I trust you are all familiar with them. You of course know what I mean when I say that an irritation in the brain leads to a hyperæmia, and to resulting disturbances in the cerebral circulation that are transient in character; for the hyperæmia is followed by a collateral œdema, corresponding to which we have symptoms of depression, while connected with the hyperæmia proper are symptoms of irritation; the collateral œdema causes an anæmia, which again may be followed by a collateral hyperæmia of other parts; and in this manner collateral œdema and hyperæmia continue to succeed each other, being evanescent in character, and consequently unable to produce symptomatic manifestations of long duration.

Cerebral tumors present three forms of *symptomatic indications*. The *first* are the symptoms of *direct and reflex irritation*. Those of *direct* irritation we may call symptoms of *excitation*; and those of *reflex* irritation, symptoms of *radiation*. Having already fully explained this point in the example of the tumor pressing on the third pair of nerves, it is not necessary to dwell upon it any longer. In the *second* place, we have symptoms of proximate results, or of *local lesions*. Suppose we have a tumor situated on the medulla oblongata,—that the tumor grows there, continually encroaching upon its constituent portions: will not this disturbance give rise to certain characteristic local manifestations? Will not the tumor produce certain histological changes, and cause more or less interference with the proper functions of the medulla oblongata? It certainly will, and the symptoms will immediately point to functional impairments of a physiological nature, the result of a pathological condition and of a local lesion. The *third* set of symptoms are those of *compression*. If there be a tumor,—an exostosis, for instance,—or an aneurismal dilatation encroaching upon the base of the brain, of course there will be a compression exercised, which may be propagated or transmitted to other parts of the brain, and attended by corresponding symptoms.

The next point of importance is the value to be attached to

the presence of *fleeting* and of *permanent* symptoms. When we diagnosticate a cerebral tumor, and have fleeting symptoms, we naturally think of the effects of irritation,—the result of recurrent hyperæmia and secondary œdema. But if the symptoms are permanent in character,—if they are those of depression, no longer evanescent, but consist of continued paralytic phenomena,—then they are no longer explicable by congestion, or referable to a transient tissue-irritation, but are the result of a constant and progressive invasion and encroachment upon some important part of the cerebral substance.

We now come to the *ordinary, general* symptoms of cerebral tumors. How do they commence? They are usually at first diffused, not pointing to mere local lesions, but evidences of widespread disturbances resulting from the fact that the entire brain feels the noxious influence of the foreign growth against which it rebels. *Cephalalgia* is commonly the initial one, and is very important. The headache is persistent, lancinating in character; it is agonizing, and almost unbearable in its prolonged duration. With the exception of meningitis, I know of no cerebral disease in which the headache is so violent. The pain may be experienced at any point of the brain; it may be frontal, vertical, or parietal, and does not necessarily correspond with the site of the tumor. Then there are certain *psychical symptoms*; also hyperæsthesia of the nerves of special sense. The mental symptoms are those of undue irritation: the patient is more or less morose, peevish, and irritable, and presents some of the signs of hyperæmia of the brain. The nervous hyperæsthesia may involve the optic nerve, or we may have an exalted sensibility referable to the auditory nerve, or to the nerves of smell or of taste; these hyperæsthesiæ may be either limited or general in character,—as a rule, however, they are limited and partial. In rare cases the symptoms of irritation are absent. I recollect a patient in whom I diagnosticated a cerebral tumor, and whose condition had been from the very commencement of his illness one of *depression*. He was very apathetic and somnolent, had had but little headache, but a paralysis of the motor oculi; he was in a state of mental hebetude,—evinced no interest in the ordinary affairs of life, although he was a husband, a parent, and a business man. Depression of spirits and mental lethargy are sometimes present instead of the more common

state of cerebral irritation ; but they are rare, except towards the termination of the malady, and are, therefore, an exception to the rule. Certain *vertiginous* symptoms appear sooner or later.

When the tumor is under the tentorium cerebelli, in the posterior cranial fossa, the vertigo is very characteristic and pathognomonic. You remember that I described its peculiarities in the early part of the course. But, whether the tumor be in one cranial fossa or another, some form of vertigo is always apt to be developed, though not always of precisely the same nature. When superinduced by disease in the posterior fossa, the patient is comfortable when quiet, but when he maintains the erect posture, or walks, the vertiginous feeling is excited, from which he is relieved only on assuming the recumbent position.

In cerebral tumors there are sometimes certain "*bizarre*" and indescribable *sensations*. The patient will tell you that he feels that there is something growing in his head ; that his skull is about to burst ; or, again, he may have a sensation as of water flowing in undulating currents to and fro, and many other such curious impressions, the existence of which is quite characteristic of certain forms of tumors. They are, however, as often absent as present.

Let us now consider the *symptoms of mesocephalic excitation*,—symptoms manifested in consequence of an irritation of the mesocephalon, which, as I told you, is the intra-cranial prolongation of the spinal medulla, and consists of the medulla oblongata, the pons varolii, the crura cerebri, etc. Whenever irritative lesions extend to these parts, there will be, almost invariably, a development of epileptiform convulsions. These are remarkably characteristic of such an occurrence, and behave exactly like ordinary epileptic attacks. You find them in the varying forms of *haut mal*, *petit mal*, and psychical epilepsy ; and in each case there is a complete loss of consciousness during the attack, although *grand mal* is most common. Convulsions occur whenever a tumor is pressing on the mesocephalon ; and just in proportion to the rapidity of its growth will there be an increase in the severity and a decrease in the intervals of the epileptiform attacks.

In a given case of convulsions there are at least four pathological conditions to be excluded before making a diagnosis of cerebral tumor. As I have lectured on convulsions, and on each one of these forms of disease, I shall only allude to them *en*

passant. They are *idiopathic epilepsy*, *lead-poisoning*, *chronic alcoholism*, and *Bright's disease*, or *uræmic convulsions*. These four pathological conditions may lead to convulsions without hemiplegia.

Vomiting, also, is a characteristic symptom of mesocephalic irritation,—not the usual gastric vomiting, but cerebral vomiting, which I explained while lecturing on meningitis; and you undoubtedly understand that I have reference to a vomiting which takes place without the least effort,—not the result of an overladen stomach or of some gastric disorder, but occurring even when the viscus is empty. It is a vomiting not preceded by nausea, and is almost invariably developed when a cerebral tumor irritates certain parts, especially at the base of the brain. These symptoms are sometimes complicated by obstinate *constipation*: this fact should never be overlooked, for, in certain cases where it is difficult to make a correct diagnosis, its presence may be of assistance.

The third set of symptoms, to which I have already alluded, and to which I shall again refer, consists of the *circumscribed, local symptoms* proceeding from the *focus* in which the tumor grows; we divide them also into *two groups*,—namely, symptoms of *excitation*, and symptoms of *depression*.

I presume you will understand that if a tumor is situated in the immediate neighborhood of the pons varolii the character of the symptoms developed in consequence of its involvement may be those of excitation or those of depression; but the manifestation of the different morbid phenomena would almost invariably indicate the pons varolii as its point of departure. Consequently, by strict anatomical considerations, and a careful observation and analysis of the different symptomatic, pathological manifestations, in connection with the disturbance of the physiological functions of certain parts, you may often arrive at a correct conclusion as to the portion of the brain involved, whether it is the medulla oblongata, the crura cerebri, the pons varolii, or some other cerebral centre.

Sometimes *febrile reaction* is developed. It is not due to the primary effect of the tumor itself, but the result of secondary irritative conditions, such as an encephalitis, or meningitis, depending on the encroachment of the tumor on the neighboring

structures or membranes, the irritating effects of which excite an inflammatory action, causing encephalitis, or meningitis of a suppurative character. Therefore, in all cases of cerebral tumors, when certain febrile phenomena manifest themselves you naturally suspect a complication of a grave inflammatory disease. As a general thing, *non-irritative lesions* produce only a *transitory* state of paralysis, simply because they do not provoke secondary inflammations. The transient symptoms are attributable to direct involvement of the ultimate constituents of nerve-tissue, leading to temporary paralysis,—temporary, because the lesion is not persistent. That permanent lesions will produce permanent symptoms is a self-evident fact. Therefore in cases of cerebral hemorrhage the patient is generally hemiplegic for life. But if in a case of cerebral tumor the paralytic phenomena are fleeting in character, it is simply because the lesions themselves, or their results, are also evanescent. One day you may have a hyperæmia, with corresponding symptoms of irritation, and another day a collateral œdema will be produced, with symptoms of depression, consisting of paralytic phenomena. Then, again, the hyperæmia may disappear, or the secondary œdema may be absorbed, and the different symptomatic indications will succeed one another, or disappear entirely, as I have pointed out at the beginning of this lecture.

The *peculiarities* of the transient paralytic states are most apparent in the paralysis of the muscles of the face, eye, or limbs: however, we sometimes find the lower limbs first paralyzed, and this is about the only exception that I know of to the rule that paraplegia is always the result of a spinal, never of a cerebral, lesion. This I also spoke of in a former lecture. We cannot always explain how this occurs, but it is a fact nevertheless that sometimes the paralysis first affects the lower limbs, not as in ordinary cerebral diseases, where the upper extremities are always first affected. Again, whether the paralysis is hemiplegic or paraplegic in character, it is never so diffused when caused by a tumor as when it results from some other cerebral pathological condition. If the tumor should always be situated on the pons varolii, we might readily explain these anomalies; but generally they are due to causes of which we know but little, and which we are at a loss to demonstrate.

Usually in cerebral tumors the *reflex* and *electrico-muscular con-*

tractility and excitability are decidedly and markedly impaired. Especially is this the case when the tumor is pressing on the *peripheral* parts of some of the cranial nerves. Aphasia—permanent or not—may be present. More or less permanent disturbances of locomotion sometimes exist, and the patient may find it extremely difficult to maintain his equilibrium.

This brings us to the consideration of the *topographical bearings* of these *lesions*. The general law of paralysis, which obtains in the vast majority of cases, is that when a lesion exists in the left cerebral hemisphere, and a paralysis results, it will be on the right side of the body; or, if the lesion be in the right hemisphere, the paralysis will be on the left side of the body. This is due to the fact of the decussation of fibres, which I have already explained. However, this is not always the case in cerebral tumors. A facial palsy may be developed on the same side as the tumor, and, at times, along with this, a hemiplegia on the opposite side. I shall try to explain this by a single illustration. Imagine a tumor in the left hemisphere, entirely within it: if we have a paralysis at all, it will be on the right side, of course, the tumor being situated above the decussation of fibres. But many cranial nerves decussate at the base of the brain, and a tumor may involve a nerve either before or after the fibres have decussated; the tumor may press on a nerve just as it emerges from the base of the brain, after it has become free, and, necessarily, the paralysis would then be on the same side as the tumor. If, then, we imagine a tumor pressing on one of the third nerves, just after its exit from the base of the brain, we will have a strabismus, ptosis, etc., on exactly the same side as the tumor; yet at the same time the tumor may be pressing more deeply on certain important parts *within*, which will cause a motor paralysis of the other side. This anatomical fact explains the want of correspondence which sometimes exists between a facial palsy and a hemiplegia. As a tumor progresses in size, we sometimes ascertain the supervention of an *impairment of the functions of some cerebral nerve*,—not a mere partial anæsthesia, but a total functional suspension. Any one or all of the nerves of special sense may be involved (but the result is usually more limited), and their functions become totally extinct. This is one of the pathological results of a cerebral tumor; and you know that we have symptoms either of irritation or of depression,

—either a hyperæsthesia or an anæsthesia. Very often we find a marked *impairment of the intellect*; still, as I have already told you, there may be no defective ideation; and whenever a tumor in the brain is rapidly developed, we generally have epileptic convulsions; and in correspondence with the violent character of the paroxysmal convulsions and their frequent recurrence are the mental impairment and the degeneration of the intellectual faculties.

We arrive now at the conclusion of the study of the symptoms. The important ones, which I recommend you to bear in mind, are very few, and I shall recapitulate them. All that I wish you to recollect are the principal features of the symptomatology, with which you most probably will have to deal in cases of cerebral tumors. They are: *cephalalgia*, *cerebral vomiting*, violent and recurring epileptiform *convulsions*, and the peculiar *fleeting* character of the *paralytic phenomena*. Another peculiarity, also, is that generally the paralytic condition *involves special cerebral nerves*, and that there is a *limitation* of the paralysis as regards the distribution of one or two cranial nerves; but usually this paralytic condition is fleeting and limited in character, especially in the early history of the case.

As regards the *diagnosis*, there are three different points to be borne in mind. I have said enough of the symptomatology and of the etiology for you to know that the first thing necessary is to ascertain with certainty, if possible, the existence of a tumor. The symptoms may be due to other forms of cerebral disease, and it is your duty to exclude them. You must then, as practical physicians, endeavor to arrive at a conclusion in regard to the anatomical site of the tumor; and this you can only do by a careful consideration of all the interferences with the physiological functions of the parts involved. As a matter of course, you must be familiar with the anatomy and physiology of the nervous system, and know every part that is presided over by the different nerves. It must be a source of great satisfaction to be able to determine the nature of a cerebral tumor,—to tell whether it is vascular, tubercular, etc. Should a scrofulous or a syphilitic cachexia exist, you will naturally ascribe the brain-symptoms to an adventitious growth due to a development of the cachexia in the brain. This will not do in cancerous tumors, which are

seldom secondary, but almost always primary, and generally have their first manifestations in the brain, without the evidence of a cancerous cachexia during the patient's life.

The *prognosis* of cerebral tumors is necessarily unfavorable: the affection being progressive in character, recovery is out of the question. Perhaps we may find an exception to this rule in the case of syphilitic tumors. Certain remedies, freely administered, —iodide of potassium, and others,—may have a decided influence on such growths, and even cause their absorption.

In regard to the *treatment*, there are two points to which I shall call your attention. Whenever a particular diathetic condition exists, and you consider the tumor to be the expression of this condition in the brain, direct your treatment to the diathesis. As I just remarked of syphilis, whenever the life of the patient is jeopardized by secondary complications, treat actively and without delay. The same thing must be done when the danger occurs from the great violence of secondary hyperæmia; and measures of local depletion, purgatives, and counter-irritants may be used to advantage. Very often the patient will die sooner from the secondary hyperæmia and collateral œdema than from the cerebral tumors themselves. You therefore understand that although the disease is progressive, and ultimately necessarily fatal, the proper diagnosis and a judicious treatment are nevertheless of great importance, for the first will guard us against complications which by the second we may properly meet, and thus perhaps be enabled to prolong the patient's life.

LECTURE XXXIV.

PROGRESSIVE LOCOMOTOR ATAXIA.

Progressive Locomotor Ataxia.—Posterior Spinal Sclerosis.—Symptoms.—Initiatory Symptoms.—Cerebral and Spinal.—Ordinary Symptoms.—Anæsthesia.—Interference with Transmission of Sensory Impressions.—Gastric Troubles.—Disturbances of Locomotion.—Peculiarities of Gait.—Invasion of Upper Extremities.—Topographical Knowledge.—Appreciation of Weights.—Tactile Powers.—Reflex Excitability and Muscular Contractility.—Ocular Troubles.—Symptoms not Constant.—Sexual Functions.—Duration of the Disease.—Symptoms in Advanced Cases.—Diagnosis.—Paraplegia.—Rheumatism.—Syphilis.—Cerebellar Disease.—Tests for Locomotor Ataxia.—Morbid Anatomy.—Pathology.—Seat of Lesion.—Exclusion of Cerebellar Disease.—Muscular Sense.—Lockhart Clarke's Discoveries.—Treatment.

GENTLEMEN,—*Progressive locomotor ataxia* is the disease which I have chosen as the subject of to-night's lecture. Being a progressive affection of the locomotor apparatus, and ataxic in character, it is really well named: still, I believe, with Dr. Hammond, that diseases ought to be designated, if possible, in relation to their pathological significance; and this one is better expressed by the appellation, as originally proposed by that distinguished neuropathologist, *progressive posterior spinal sclerosis*: for the reason that it is a progressive affection of the posterior columns of the spinal cord superinduced by a pathological condition known as sclerosis.

As regards the etiology of locomotor ataxia there is not much to say, as we know very little about it: it has been ascribed to the syphilitic poison, also to an excess of sexual indulgence,—in short, to all varieties of dissipation and venery; but about the precise mode of causation we are positively in ignorance. One thing is certain, however, that males suffer in a much greater proportion than females; indeed, females are but exceptionally attacked.

The symptoms of progressive locomotor ataxia are quite characteristic, even, sometimes, in the initiatory stage. It is upon

these initiatory symptoms that I shall dwell first. Their being often *cerebral* in character has led Dr. Hammond to doubt whether the disease should be classed under the affections of the cerebro-spinal or simply of the spinal system; but in point of fact the presence of the cerebral symptoms depends upon the location of the sclerosis, whether it be in the upper or in the lower portion of the cord. Whenever the disease extends to its intra-cranial prolongation we have epileptiform symptoms, vertigo, and also certain ocular troubles, such as amblyopia, amaurosis, diplopia, ptosis, internal and external strabismus: certain nerves, therefore, especially the optic, third, sixth, and auditory, are very liable to be affected. When the initial symptoms are spinal in character, there is always a feeling of pain in the particular portion of the cord: it is quite peculiar, and characteristic of locomotor ataxia. The pain is darting, electric, and lancinating: the word "electric" probably conveys the best impression of it to the mind: it shoots out, with lightning-like rapidity, from the spinal region towards the four extremities. At times it is only felt in the direction of one extremity, at other times in all of them; but in all cases it is agonizing, though transient in character, and if not rapidly relieved becomes really unbearable: the relief of this pain is, therefore, a very important point in the treatment of the affection. A feeling of constriction around the waist, a symptom so common in many varieties of spinal disease, is sometimes perceived.

I have now described two sets of initiatory symptoms,—the cerebral and the spinal. When we come to study the pathology of the disease, you will readily understand why it is that the patient is so prone to the manifestation of symptoms pointing to a disturbance of co-ordination in the voluntary muscles: you will also comprehend the different varieties and grades of *anæsthesia* which we so often meet in locomotor ataxia. For instance, when the sclerosis affects the lumbar portions of the cord, we have a plantar anæsthesia; and where the part affected is in the cervical region, there is a palmar anæsthesia. While considering the different and significant facts connected with anæsthesia, we shall do well to study, first, the change in the *transmission of sensory impressions*. The transmission is always interfered with so far as its normal rapidity is concerned; and sometimes the propagation of the sensory impressions is entirely arrested. To make this plain

I shall have recourse to an illustration. Suppose I touch one of you: the very moment I do so you will recognize the fact; but if I touch an individual suffering from progressive locomotor ataxia, several minutes may elapse before he becomes aware of it: this is on account of the loss of normal rapidity in the transmission of the sensory impressions. This impairment may exist in different ways, and the patient may appreciate one or more of the various perceptions which are embraced in sensation, and not others. He may appreciate temperature and not pain; you may prick him with a pin and he will not feel pain, but he will readily discern a change in temperature; the reverse may also exist; or you may pinch him without producing any sensation, but the sensation of tickling will perhaps readily be distinguished, etc. These peculiarities have been explained by Brown-Séquard, who has shown that in each centripetal sensiferous nerve four distinct fibres exist, transmitting four distinct kinds of sensation, one appropriate fibre to each sensation. We have, then, one fibre transmitting the sensation of touch, one for the feeling of tickling, one for the appreciation of temperature, and one for the perception of pain. These fibres, although existing in close apposition, are nevertheless entirely individual and distinct, independent of one another; so that one may be pathologically implicated and the functions of the others not be interfered with; or several or even all of them may be involved; and then the transmission of all of the sensations will be entirely arrested. So tardily are impressions sometimes conveyed, that Dr. Hammond cites the case of a person suffering from locomotor ataxia whose feet could be plunged into hot water without the individual experiencing any sensation for several minutes,—at least he would not realize the fact for an appreciable interval of time: of course, if his feet had been allowed to remain in the water under such circumstances, they would have been thoroughly scalded and the superficial tissues destroyed. You now undoubtedly understand how the transmission of sensory impressions may be interfered with or retarded.

Certain marked symptoms are always found in ataxia whenever there is marked anæsthesia of the lower extremities. The patient feels as if his shoes were too long, or as if he were walking on feathers; sometimes there is a sensation as if some object were

interposed between the sole of the shoe and the stocking. I recollect a gentleman who had suffered from ataxia, whom I asked if he ever felt this strange sensation : he replied that he had experienced it so persistently that he had often taken off his shoes to convince himself whether the feeling was or was not correct. The diversity in these perverted sensations is very great : in his article on locomotor ataxia Dr. Hammond speaks of a person who complained that he felt as if his feet had been immersed in tar and then sand had been dusted thereon : this feeling was most evident in walking ; again, such people often have sensations of formication, as if insects were crawling over them, or as if pins and needles were pricking them constantly. This last, however, is common in certain anæsthetic disturbances in several other diseases.

As the disease progresses, very often *gastric* and *intestinal* troubles manifest themselves, and are frequently ascribed to dyspepsia ; these gastro-enteric complications are sometimes so serious, and their symptoms so prominent, that physicians not infrequently overlook the true nature of the disease and treat ataxia for dyspepsia.

We shall now consider the most important, interesting, and distinctive symptoms of progressive locomotor ataxia, namely, the *disturbances of locomotion*,—the pathognomonic feature of the disease. When a man walks, he does so in consequence of the particular function of those nerves which preside over the co-ordination of muscular movements ; for in order to walk, or to perform any ordinary action which calls into play the working of several sets of muscles, it is evident that the movements must be regulated with precision, that there should be no confusion, that one set of muscles should contract exactly as the other set relax : in short, the regularity of the antagonistic action of muscular contraction and relaxation constitutes the co-ordination of voluntary muscular movements ; and without this co-ordination the proper execution of certain fine and complex muscular actions is impossible. When studying the pathology of the disease, we shall ascertain what causes the want of co-ordination : as it is, the patient becomes conscious of his inability to walk in a normal manner, and loses confidence in his own powers of locomotion ; we then have the characteristic gait, which forms the most prominent symptom in locomotor ataxia.

The patient being uncertain of his movements, and no longer able to co-ordinate them, he staggers like a drunken man at every attempt to walk; if the disease be still in the early stage, anything that will inspire confidence will facilitate his progression, and he will do quite well if you guide him by letting him take hold of your hand, when, at the same time, he calls into aid the powers of vision, fixing his eye upon the object to which he desires to walk. But if you deprive him of this help he will be powerless and unable to advance. Now, I do not want you to misunderstand me, and to imagine that the disease is paralytic or paraplegic in character; for such is not the case. There is no loss of motor or muscular *power*, but simply a disturbance of the voluntary movements, in consequence of the absence of the necessary co-ordinating influence: the patient may give you as decided a kick, or make as energetic a movement, as when in perfect health; he may even, if you desire it, carry you on his shoulders, if he is a muscular man, only the movements will not be co-ordinated and regular, and will be more fatiguing than would be the case if the co-ordinating nervous centres were not invaded by the disease. The gait, therefore, is the characteristic of the disease, and it is often the first symptom that will attract the patient's attention, especially if there is a coincident development of the electric-like pains.

Sometimes the first intimation the patient receives is a peculiar dizziness while shutting his eyes; and this may occur while performing certain customary acts. Hammond relates a case, very interesting, of an individual who was made aware of this disease by being in danger of falling from staggering immediately when he shut his eyes during his morning ablutions. This was the first evidence of the disease, and is enough to strike an experienced physician.

The reason why the sense of sight often enables an ataxic person to walk is, that when one organ is weak or deficient we call for additional or compensatory assistance upon another not affected; and it is by concentrating the vision upon an object, and not losing sight of it for a moment, that such an individual is sometimes enabled to approach it; whereas if you were to ask him to shut his eyes he would immediately stagger and fall. But, independently of the difficulty in walking, *the gait itself is peculiar*, and

a pathognomonic symptom of locomotor ataxia. It is so striking that any experienced physician can readily single out an ataxic patient simply by seeing him walk.

In the normal gait the heel strikes the ground first, and then the ball of the foot comes down; there is an interval, but the two motions are so nearly simultaneous that it is almost inappreciable. In locomotor ataxia there is an interval of longer duration, which may perhaps be detected even by the ear. The patient's foot does not describe a semicircular line, as in hemiplegia, but is thrown forward by a peculiar spasmodic action; then the heel is brought down with great violence, and after an appreciable interval the ball of the foot strikes the ground.

Sometimes, in consequence of a plantar anæsthesia, the patient is unable to place his foot upon particular small surfaces; he cannot adjust it to a stirrup, or on the step of a carriage, etc.; or to direct it to any small space seems to be a source of great difficulty,—in most cases quite a significant symptom.

As regards the peculiarities of gait, the foot not only strikes the ground with violence, but, in consequence of the patient's want of confidence, he invariably widens his base for additional support. Instead of throwing the feet directly forward, he throws them obliquely, first to one side, then to the other, resembling somewhat the behavior of a drunken person trying to maintain his equilibrium. In locomotor ataxia the walk is so characteristic as to be readily recognized, and if you notice ataxic persons critically you will find the widening of the base so evident as to be quite striking.

When the *upper extremities* are invaded in locomotor ataxia, the hand is more particularly liable to be affected, and the patient will no longer be able to perform any of the finer muscular movements with the precision of which the hands and fingers especially are normally capable: he will find it impossible to tie a cravat, to button his coat, or to thread a needle; if he attempts to pick up a pin he will, perhaps, succeed with great difficulty, and if he tries to bring a fluid to his lips he will not fail to spill some of it: all in consequence of his being deficient in the power of co-ordination.

A very striking symptom is the faulty appreciation of *topographical points* by the patient. Any of us may close our eyes

and still be able to touch any part of the body desired; we may make a mark on our forehead the size of a pin's head and readily touch it; we can easily put a finger on the tip of either ear, or on an incisor tooth, or on the tip of the nose. We do this by a certain topographical sense acquired by habit and education and dependent for its regulation upon the spinal centres. But in locomotor ataxia it is quite different: you ask such a patient to touch the tip of his nose with his finger, and in his efforts he is just as liable to put it on his ear; or you ask him to bring his hand to his mouth, and perhaps he will put it on the top of his head: in fact, he is at a loss to appreciate the topographical location, owing to the disease.

Recollect that more commonly it is the lumbar portion of the spinal cord and its peripheral nerves which are affected, and then the ataxic manifestations are in the lower portions of the body; when the upper extremities are involved, the trouble is in the cervico-dorsal region; and when cerebral or ocular symptoms are present, the intra-cranial prolongation is invaded.

Another symptom of progressive locomotor ataxia is the difficulty in the *appreciation of weights and measures*. When a man in normal health places two bodies of different weights, say one of eight pounds and one of nine pounds, in either hand, he can readily determine which is the heavier of the two; but when an ataxic patient has an involvement of the upper extremities, he cannot readily appreciate this difference: this symptom is considered quite an important one by many authors. There is also a diminution of the *tactile powers*: this is readily measured by the *æsthesiometer*, but in the absence of an instrument it can be quite as well determined by an ordinary hair-pin. If I were to take a compass and open its two arms about an inch apart, and then press the two points against any part of your body, you would immediately recognize two distinct impressions; but an ataxic person might feel only one, according to the greater or less separation of the compass-points; so that we can measure this defect with almost mathematical correctness. I should state, however, that this occurs not only in locomotor ataxia, but also in other forms of spinal disease.

Another important symptom is related to the *reflex excitability* and the *electro-muscular contractility*. In locomotor ataxia these are always decidedly increased and exaggerated. Another symptom

of which I have spoken as present in certain spinal diseases is also found in locomotor ataxia,—namely, the sensation of a *cord tied around the body*.

As regards the *ocular troubles*, I have referred to them at the beginning of this lecture. I told you that it is particularly the optic, third, sixth, and auditory nerves which are involved. These are significant facts as regards the diagnosis.

Another peculiarity of the symptoms is that they are *not constant*, but are fleeting in character; for instance, certain symptoms which generally exist in the early part of the affection often disappear as the disease progresses. It also bears a certain relation to the *sexual* functions. As a general thing, they are much increased in the earlier stages, but in the later, impotency is developed. It is not uncommon to hear persons suffering from locomotor ataxia, early in the attack, state that they are unable to gratify sufficiently their sexual desires,—that they perform the act of coition several times in one night, and still without appeasing their venereal appetite. Some complain of being much weakened by nocturnal seminal emissions: this is very common. On the other hand, you will find ataxic patients in whom the sexual appetite is entirely extinct, they being incapable of having an erection of the penis.

We shall now proceed to consider the *duration of the disease*. All I can say about it is that it is very variable and not subject to any limitation. Jaccoud says it runs its course in from six to eight years; but many authors claim that the limit cannot be computed, being variable,—that it may last ten, fifteen, or twenty years, and that its termination may be deferred until some inter-current disease eventually cuts off the patient.

As the *disease advances*, the *symptoms* become more manifest and hopeless, and the initiatory locomotor disturbances gradually pass into an entire abolition of the powers of locomotion. The results of the immobility to which some of the voluntary muscles are condemned are several: of course, after a certain length of time they become atrophied; the impossibility of walking keeps the patient in one position, and produces a great tendency to the formation of bed-sores, and to an inflammatory condition of the joints. The disease was often confounded with rheumatism before progressive locomotor ataxia was described as a distinctive affec-

tion. The late Professor J. K. Mitchell, of Philadelphia, cited cases of spinal affection that showed a remarkable connection with an inflammatory condition of the joints. Some of the cases he referred to were evidently instances of locomotor ataxia.

We have now reached the most important point: it is the *diagnosis*. What diseases may locomotor ataxia be confounded with? The one which is generally mistaken for it is *paraplegia*.

Trousseau says that in the Paris hospitals many persons were formerly considered hopelessly paraplegic when they were actually suffering from locomotor ataxia; and in some instances, to convince the hospital physicians of their error, he placed himself on the shoulders of an ataxic individual and allowed himself to be carried around the ward. This was proof conclusive, for Trousseau was quite a corpulent person; and, had the patients suffered from myelitis, it would have been an utter impossibility for them to walk at all. In such cases there was no paraplegic trouble, no involvement of the power of motility, no disease of the antero-lateral columns of the cord,—but simply a want of co-ordination of muscular movements, causing the patient's walk to be disordered. When a man is drunk, he cannot maintain his equilibrium; he staggers and stumbles, and often keeps himself erect only by grasping a neighboring object; but, if he is pushed to do so by some cause or other, he may administer a powerful kick, or even, getting a fair start, may run away. It is much the same in locomotor ataxia; and this is a distinctive feature between this affection and myelitis, or other paraplegic troubles: these, therefore, ought not to be confounded; and if you pay any attention to the history of the case and its principal features, it will be almost impossible for you to commit such an error.

Now, strange as it may seem, it is frequently mistaken for rheumatism. The sclerosis produces the lancinating pains already described, and certain physicians, prone to jump at conclusions, immediately pronounce it a case of rheumatism; the disease advancing, the joints become swollen, which, to their minds, is corroborative proof of the correctness of their diagnosis, having pronounced years before that the person was afflicted with rheumatism. But, by ordinary discretion, and by not overlooking the nervous symptoms, a careful physician will readily diagnosticate correctly, and ascribe the painful swelling, not to a diathetic

rheumatic condition, not to *materies morbi* of any kind, but simply to a locomotor ataxia, or, what is identical in meaning, a sclerosis of the posterior columns of the spinal cord.

Syphilis has certain bearings on locomotor ataxia, as well as on nearly all nervous diseases. The fact is that syphilis is so common and so important, and when it affects a patient may present so many protean symptoms, especially when it invades the domain of the nervous system, that it is not at all impossible, or even improbable, that it not infrequently enters as a factor in the disease which at present occupies our attention. I therefore advise you, in all cases of ataxia where a history of syphilis is given, or where evidences of syphilis, however slight, are present, to use antisyphilitic measures, and oftentimes the patient will recover. As I just said, so many nervous diseases are of syphilitic origin, that whenever you have the slightest evidence of syphilis, whether in diseases of the cord, of the brain, or of the peripheral nerves, you should always have recourse to an energetic antisyphilitic treatment.

The next question is this: Is locomotor ataxia, as is often taught, and as was formerly held by Duchenne (who was the first to describe the disease, and by whose name it is still known), is ataxia due to a *cerebellar disease*? In answer to this, suffice it to say that in a post-mortem examination after uncomplicated locomotor ataxia you cannot find any evidence of disease of the cerebellum; moreover, the symptoms during life are quite different from those of that affection. In this last you have a peculiar vertigo and occipital pain, with vomiting; also an impaired articulation and deglutition, and a difficulty in walking, we will admit; but this inability to walk is the result of the vertiginous sensation: it is not dependent upon a want of co-ordination, but is caused by dizziness, which is always accompanied by localized pain and is characteristic of cerebellar disease. Modern physiology, and especially the experiments of Hammond, have, to my mind, proven conclusively that the cerebellum is not, as was formerly taught, the organ which presides over the faculty of co-ordination. Its ablation, even, has not produced permanent effects upon muscular co-ordination, provided the animal recovers. Pathological observations of others are also confirmatory of the correctness of Dr. Hammond's conclusions. Here, again, if you

observe carefully the clinical phenomena in ataxia, no error is possible. Malaria sometimes produces functional disturbances very similar to those of locomotor ataxia, which readily yield to iron, quinine, and arsenic.

The next thing to consider is, whether there are any particular *tests* by which we can always ascertain the existence of locomotor ataxia. There are two which you will find of clinical importance, and to which Dr. Robinson has undoubtedly called your attention during the clinics at the city hospital. The *first* is, when you are in doubt, order the patient to approximate the internal borders of his feet, and then to close his eyes. If he suffers from ataxia, he will immediately stagger, and, if not supported, will fall. The *second* is, let the patient walk towards you with his eyes closed. This, also, he will be unable to accomplish.

I have once before explained how it is that these patients in an advanced stage sometimes manage to walk after a peculiar fashion only when they have called to their aid the organs of vision. So, whenever it happens that a person comes to you and states that when he arises in the night to urinate, the room being dark, he immediately staggers and sometimes falls, always suspect locomotor ataxia, and do not fail to examine him critically with reference to its existence.

We shall next consider the *morbid anatomy* of progressive locomotor ataxia. The morbid anatomy consists in a sclerosis of the posterior columns of the spinal cord. I have used the word sclerosis several times, and its meaning is perhaps enigmatical to most of you; but I shall proceed to explain. A sclerosis, in its most simple sense, is an increased proliferation of the cerebro-spinal connective tissue, the neuroglia of Virchow, the cement, as it were, binding together the fibres and cells. If in sclerosis, then, we have such a proliferation of tissue-corpuscles, it must necessarily be at the expense of the ganglionic nerve-cells and other histological constituents. Something is bound to give way before the encroachment, and in this case it will be the nerve-fibres and ganglionic cells, which will atrophy and ultimately disappear; and, as a direct result of their atrophy and disappearance, we shall have, sooner or later, an impairment or a total abolition of their physiological functions.

The next question which presents itself is the *pathology* of

locomotor ataxia. It has always been taught that the faculty of co-ordination of voluntary muscular movements was at fault; but this actually tells us little or nothing. But what causes it? There must evidently be an involvement of those nerve-cells which preside over the co-ordination of the muscular movements. But where is the lesion situated? Duchenne, the first pathologist who studied and described the disease, maintained that the *seat of lesion* was the *cerebellum*; but he afterwards recanted, and repudiated this idea. I have told you already that in a post-mortem examination no traces of cerebellar lesion can be found, but we do find lesions in the posterior columns of the cord: hence we are right in concluding that locomotor ataxia is *an organic disease of these posterior columns*.

Now, what does physiology teach? It teaches us, as I have before stated, that we may extirpate the cerebellum from an animal, that this will first produce a shock and vertiginous manifestations, but that, if the animal does not perish, the vertigo ultimately passes away, the animal will no longer stumble, but will walk as well as before the operation. This proves beyond a doubt that the cerebellum does *not* preside over the co-ordination of muscular movements. Neither does the cerebellum preside over the *sexual functions*, as was formerly claimed. Flint, in his "Physiology," states that the cerebellum of the cock may be extirpated, and still, if he survives, he will tread the hen as before, and his erotic faculties will not evince any impairment. If we study Andral, who treated and collated a large number of cases of diseases of the cerebellum, we shall learn that of ninety-three instances the want of co-ordination over the voluntary muscular movements was impaired only in one single case.

But it has been contended by certain authors that there is such a thing as a *muscular sense*; that it is necessary to appreciate certain *sensory impressions* in order to perform the different muscular movements correctly; that the former are conveyed from the muscles to the brain, and from the brain to the muscles; that the sensations are transmitted upwards and then down again by an imaginary circle of action; but Trousseau has conclusively proven that there is no muscular sense, as I shall now explain. If we tell an intelligent person, a person of education, but ignorant of the laws of physiology and of anatomy, to grasp this railing, and

then ask him what muscles he contracts, he will tell you those of the hand; but this will not be correct: it is the muscles of the fore-arm that are contracted. Hence sensations in this respect are not trustworthy.

This brings us to the consideration of the location of the powers of co-ordination. The scientific researches of Lockhart Clarke have enabled him to ascertain, among other facts, that the old doctrine that the posterior roots of the spinal nerves are attached to the lateral columns of the cord is an anatomical error: he has demonstrated that these roots are attached to the posterior columns only, and have nothing in common with the lateral columns, but that there are three rootlets which perforate the gray substance, some finally reaching both the lateral and anterior columns, the remainder of the fibres losing themselves in the gray marrow of the posterior column. The ganglionic substance in the cord has nothing physiologically in common with the posterior columns, which are composed only of white fibres and convey sensation; but deep in the cord is the gray matter, rich in ganglionic cells: their function is not the transmission of sensation, but the *production* of nerve-force, therefore they are always connected with excito-motor or reflex action. The gray matter of the cord sends out impulses of an involuntary character, and the white nervous fibres transmit or conduct it. It is, therefore, along the small rootlets that the power of co-ordination is propagated from the gray substance in which it is elaborated, and therefore the posterior columns are connected with the transmission of co-ordinating influences of muscular movements, as well as the conveyance of sensations from the periphery. This has been clearly proven by Hammond. If, then, there is a disease of the posterior columns, there must be an involvement of some if not of all of these three rootlets, and the one involved cannot transmit the impulse from the gray matter; consequently, the co-ordination of muscular movements will be faulty. So in progressive locomotor ataxia there is sclerosis of the posterior spinal columns, which may not reach the gray substance, but will surely involve one, two, or perhaps all three rootlets which are connected with the gray matter; and if all are involved in a pathological process, the proper mandates will no longer be transmitted to the muscles over which they preside, and, as a consequence, the particular

functions of these muscles can no longer be properly performed and regulated, though the motor power itself may not be interfered with in the least. The posterior columns of the cord are composed of white substance and conduct sensation: if they be sclerosed, their power of conduction will be interfered with, producing an anæsthesia the development of which will be in proportion to the extent of the sclerosis itself. I believe this is the entire philosophy of the question.

We shall now conclude the subject by considering the *treatment*. It has been recommended by high authorities, especially Hammond, to make use, first, of the ordinary galvanic or continuous current, applied to the spinal region and to the sympathetic system. The next thing recommended is the administration of bromide of potassium or of ergot. It may be that these will have a certain influence over the morbid proliferation in the earlier stages; but as the disease progresses, and after the initiatory symptoms have passed away, I have great doubts of their efficiency. Nitrate of silver has also been recommended, and is held in high esteem by some; but, notwithstanding it enjoys some reputation, I must confess that where we have an organic disease of either the brain or the spinal cord my faith in remedies is exceedingly weak. The affection is progressive in character, and therapeutic measures have generally but little effect on progressive pathological processes. Still, as you must do something, you might try the bromide of potassium with ergot first, and nitrate of silver afterwards. It is my opinion, however, that when the patient is debilitated the administration of phosphorus and of fatty or oleaginous substances, such as the pancreatic emulsion, or cod-liver oil, is often very efficient in toning up the system and improving the general condition. If the slightest suspicion of syphilis exists, an antisyphilitic treatment is indicated with fair prospects of success.

LECTURE XXXV.

DIPHTHERITIC PARALYSIS.

Mild and Severe Forms.—Mild Form.—Symptoms.—Severe Form.—Diagnosis.—Mutability of Symptoms.—Old View of Pathology.—Albuminuria.—Pathology.—Prognosis.—Treatment.

GENTLEMEN,—*Diphtheritic paralysis* is a form of disease which, though not of recent origin, has, nevertheless, only been described and recognized during the present century. Just as in scarlet fever there is a liability to the development of certain sequelæ, such as otorrhœa and albuminuria, or as in measles there is often a supervention of bronchitis or of lobular pneumonia, so is diphtheria sometimes followed by certain paralytic phenomena, which it behooves you to recognize and familiarize yourselves with, in order to appreciate their importance when they occur, and especially in view of their prognosis and treatment.

The reason why diphtheritic paralysis is now more frequently met with than formerly is not because it really occurs oftener, but is simply due to the fact that the connection which exists between diphtheria and certain forms of paralysis is better understood than was formerly the case. If a physician is familiar with the disease, and the conditions under which it is most liable to appear, he will watch for it, and be continually on the *qui vive*. Why is it that we so often hear of albuminuria nowadays? or why do we have so many cases of leucocythæmia, which is a pathological state, only recently described by Bennett and Virchow? Is it not simply because, under certain circumstances, physicians now anticipate them, and not because the diseases did not exist heretofore? Remember that medicine is a progressive science, and that in consequence of its development several varieties of diseases are at present more easily recognized, knowing, as we do, their peculiarities and characteristics, and the circumstances most likely to produce them; and this is exactly the case in diphtheritic paralysis.

This disease is susceptible of division into *two distinct forms*, a severe and a mild form. The *severe* or adynamic or malignant form is a disease of great gravity, and generally carries the patient off with alarming swiftness. This malignant type bears the same relation to diphtheritic paralysis that it does to all other diseases, and the meaning of the word itself almost necessarily presupposes a fatal termination. The *mild* form, which I shall first describe, sometimes manifests itself during the period of convalescence from diphtheria, and generally about three or four weeks after the patient has commenced to recover,—just about the time the attending physician thinks he has the patient completely out of danger, and is congratulating himself upon the result. In some cases the first symptoms of diphtheritic paralysis show themselves very shortly after the disappearance of the diphtheria itself; in others the paralytic phenomena occur even before the disappearance of the diphtheritic sore throat; but in the great majority of instances it is when about three or four weeks have elapsed since the recovery that the symptoms of the disease are first noticed.

Following some cases of diphtheria, if the physician be on the alert, the first thing that will attract his attention is the fact that the patient has a nasal intonation affecting his speech. This peculiar nasal character of the voice, entirely unnatural to the patient, is generally the first note of alarm which strikes the attentive observer. Knowing, as he does, the peculiarities of the paralytic phenomena in this affection, he of course expects the velum palati to be paralyzed, for the soft palate is usually affected prior to the involvement of other parts of the body. In consequence of this paralysis of the velum palati there is not only a nasal intonation of the voice, but there is also a marked tendency to the regurgitation of food when an attempt is made to swallow it: sometimes, according to the nature of the food, it is even projected into the nostrils. The regurgitation is more apt to occur with liquids than with solids; but the peculiarity about swallowing solid food is that the difficulty is in proportion to the smallness of its bulk; that is, a small alimentary bolus is swallowed with more trouble than a large one. In consequence of this there is a veritable *dysphagia*, and the patient actually dreads the taking of food, for it is a source of irritation and also of danger. The food, instead of being conveyed to the œsophagus, where it should go, is some-

times forced into the respiratory tract, into the trachea; and it has happened that patients affected with diphtheritic paralysis have died of suffocation while eating, a part of the food having blocked the respiratory tube. Fortunately, however, death is not often produced in this manner. In the early stage of the disease, the velum palati, the muscles of the soft palate, and those forming the pillars of the fauces are almost invariably affected; but as the disease progresses the paralysis may extend to the true pharyngeal muscles, to the constrictors; the difficulty of deglutition will then be more marked, and if the latter refuse to perform their physiological action, if they no longer propel the food downwards to the stomach, a terrible state of affairs may ensue, and the patient's life become seriously endangered.

Now, do not forget that you may look for the appearance of diphtheritic paralysis about one month after the disappearance of the diphtheritic angina; but do not take it for granted that this is the exact time, and that the latter is invariably followed by this affection. Neither should you suppose that this is a new disease: it has probably existed as long as diphtheria itself, which, on account of its ancient origin and its prevalence in a certain country, the French call the "Egyptian disease." Another thing that you should consider is, that the gravity of the paralytic phenomena in diphtheritic paralysis bears no relation whatever to the duration or the gravity of the previous disease, the *angina pellicularis*, or pseudo-membranous sore throat; neither is there any connection between the paralysis and the albuminuria which sometimes complicates cases of diphtheria. I shall revert to this and explain it in a few moments. For the present, simply bear in mind that although when the paralytic phenomena are developed albuminuria sometimes co-exists, and although some writers claim a relationship between this combination of pathological phenomena, still, it exerts no influence as of cause to effect, as I shall subsequently prove.

But let us come back to the symptomatology. I cannot give you an exact or typical description to which all cases will closely correspond, but I shall detail to you the most ordinary symptoms in the order in which they usually follow one another. Now, the initiatory symptoms of diphtheritic paralysis I have just discussed; but, as the disease progresses, other parts may become affected:

for instance, a paralysis may be developed in the orbicularis oris, and, of course, the patient will no longer be able to perform certain actions which require the motion of this muscle; he cannot blow out a candle, or whistle, or suck, or smoke,—the physiological function of the muscle being lost in consequence of the nervous influence of the diphtheritic blood-poison.

If you look at the throat, as in all cases you should invariably do, you will find that the velum palati hangs down, occluding the pharyngeal space, and you cannot see the posterior pillars of the fauces or the posterior portion of the pharynx. If now you touch the velum palati by means of a probe or a long needle, you will discover that it is in a state of complete anæsthesia; pricking or tickling it will not produce the distressing feeling of nausea which would so readily supervene if the throat were in its normal condition. There is always in health a decided intolerance in the velum palati to manipulation, and you can convince yourselves of this by experimenting on your own throats: a very disagreeable and uncomfortable sensation will be the result; but in diphtheritic paralysis this manipulation can be resorted to with impunity. Again, when the velum palati is affected, if you observe it carefully you will notice that it does not have the ordinary oscillatory motion, the alternate retraction and relaxation, which it possesses in health; the curtain is in a state of immobility, and remains dropped; no external influences affect it. The phenomena may be limited in character,—the paralysis may be restricted to the velum palati and not extend to other parts; or, as happened in a child under my own treatment, the muscles of the eye may alone be affected, and a strabismus be the result, without concomitant symptoms. Now, in this case, had I not known that the child had previously suffered from diphtheria, and that this disease may be followed by ocular troubles, I might have been at a loss to determine the nature of the trouble I had to treat; but in diphtheritic paralysis, as in locomotor ataxia, there is a certain tendency to the production of visual troubles,—the result of paralysis of the muscles of the eye. But, to repeat, when the paralysis is partial and restricted, when there is no disposition to a general paralytic affection, it is generally the velum palati which is singled out. In this, as in almost all the progressive forms of paralysis (and diphtheritic paralysis is pro-

gressive in character, for all the muscles of the body may become involved), there is commonly a development of certain marked forms of anæsthesia. Complete anæsthesia may be preceded by these phenomena, which are the ordinary precursors of anæsthesia, no matter what the disease may be which produces it, whether a myelitis, an ataxia, or something else. The precursory phenomena are a numbness in those parts in which it is to be located, or the sensation of insects crawling over the skin, called formication, or the feeling of pins and needles. Again, as the anæsthesia becomes more marked and developed in the lower extremities the patient when walking will not feel his shoes, neither will he feel the ground: it will seem to him as if he were walking on feathers, or on something that gives way and is soft and insecure under the foot.

This may often furnish you the clue to subsequent manifestations, for the lower extremities are usually affected before the upper ones, and the motor paralysis is generally preceded by sensory disorders. In connection with the paralysis of a limb you often find an analgesia: by this is meant an absence of pain, so that a person may be pinched or bruised and still will not feel any pain. The analgesia may be permanent, or it may be transient. Some of you, perhaps, may not understand the difference between *analgesia* and *anæsthesia*. Analgesia is only a form of anæsthesia: it relates to an anæsthesia of those nerve-fibres which transmit the sensation of pain. You recollect that I spoke in my last lecture of four different fibres intimately connected, and told you that each of those fibres propagates a distinct and different sensation,—one for pain, one for tickling, one for touch, and one for temperature. Each of these may be separately affected. Now, when any fibre no longer transmits its appropriate sensation there is an anæsthesia, which, when it affects particularly the fibre transmitting the feeling of pain, we call an analgesia. I hope this is plain enough. Another peculiarity of the analgesiæ, or of the different forms of anæsthesia, is that they are limited to certain regions,—a feature characteristic also of hysteria. When speaking of the latter, I told you that the hyperæsthesia which often exists has certain particular lurking-places, certain regions which it affects in preference, and that, for instance, if you touch a hysterical woman under the left breast the pain produced may

be so excessive that she will not be able to bear it, whereas if you touch the corresponding part on the other side no such sensation will be perceived. So it is in diphtheritic paralysis: there is always a tendency to a restriction of the analgesic symptoms to certain limited regions of the body.

Now, if the paralysis extends at all, it generally affects the lower limbs, and a difficulty in walking is first noticed; there is more or less paraplegia. But the tendency is always to affect one leg to the exclusion of the other: this happens at least in the vast majority of cases. Sometimes, however, not only both legs are affected, but every voluntary muscle in the entire body is involved. As the impairment of sensation usually affects the lower extremities first, so these parts are generally first involved in the motor disturbances; and both of these may extend upwards, attacking the upper extremities. Accompanying the motor disturbances there is generally a peculiar muscular tremor; this is often best shown when the paralysis affects the upper limbs. In some instances several of the muscles of the neck are paralyzed; in this case the phenomena resemble those of progressive atrophy of these particular muscles: when the posterior muscles are affected, the head drops forward and downward; if, however, the anterior muscles are involved, the head is drawn backward or to one side.

The muscles of organic life are often paralyzed, also the sphincters, and the intercostal and diaphragmatic muscles. Now, when the patient is afflicted with paralysis of the intercostal muscles, or those of the diaphragm, if some intercurrent disease happens to complicate the case, like pneumonia or bronchitis, he is evidently in imminent danger. You easily understand that such a condition precludes the possibility of coughing or expectorating; this last act especially requires the aid of the respiratory muscles.

This brings us to the consideration of another question. I told you that in locomotor ataxia you sometimes find certain anaphrodisiac symptoms; and this is sometimes the case in diphtheritic paralysis. There is a deficiency of virility, a difficulty in the act of copulation, and often no sexual desires whatever: indeed, if the patient had these desires he would not be able to gratify them. This feature, however, is not limited to diphtheritic paralysis, but is characteristic of several nervous diseases.

The next thing to be noted is an involvement of the nerves of special sense. There may be an impairment of the nerves of smell, or of taste, or of hearing, etc.; but what is most common and most often met with in diphtheritic paralysis, whether any other nerve of special sense is or is not involved, is an affection of the *optic nerve*, and also of the *muscles of the eyeball*. Sometimes there is a marked and constant amaurosis; the patient may become entirely blind; or there may be a strabismus, or ptosis, in consequence either of a paralysis of certain ocular muscles or of a want of co-ordination in the proper movements of the muscles. Now, if you understand these phenomena, and are familiar with them, you will be able to ascribe to them their proper importance should you meet with them in practice.

This brings us to the description of the *severe* form of diphtheritic paralysis. So far I have spoken only of the mild form, in which there are certain sensory affections, as well as certain paralytic phenomena, but in which there is always a tendency to recovery. In the severe form the phenomena are quite different; the tendency is not to recovery, but to the development of convulsions, of delirium, and of coma: this form is really adynamic in character, and the system is in a state of complete prostration. It may also happen that the nervous centres presiding over the function of respiration become involved, and then there will not only be a great difficulty in breathing, but the patient will also be in imminent danger of death from apnoea.

Constituting the severe form of diphtheritic paralysis, you have, therefore, a tendency to convulsions, an impairment of the respiratory actions, a supervention of delirium, and the development of coma, *plus* the paralytic phenomena already described. All these troubles, in connection with the general depressed condition of the system, are more than sufficient to insure a fatal termination. So much for the malignant form.

You have seen that in both varieties there may be a paralysis of the velum palati, followed by a nasal intonation of voice, and also a paralysis of different voluntary muscles of the body. If an anæsthesia is manifested, it is less profound than when this phenomenon follows an organic disease of any part of the spinal cord.

The next point is the *diagnosis*; and I should not be at all surprised to hear you ask me, How are we to know this disease?

you have spoken of so many varieties and different forms of paralysis that we are often puzzled. What are the principal features, the familiar points, by which we may be able to recognize the affection? Now, the first thing is always to bear in mind that the disease follows diphtheria; were it to succeed typhoid fever or alcoholism, it would no longer be a diphtheritic paralysis: therefore here, as in all cases, the history of the case is important, and should not be overlooked. In fact, without the history of the case a diagnosis would be almost guess-work. A case of diphtheritic paralysis might be brought before us to-night, and, without the knowledge on our part of a previous diphtheritic attack, we might be unable to make a correct diagnosis, unless we possessed some clue which might put us on the right track. Simply taking the indications furnished by the sensory impairment, or the disturbances of motility, would surely not be sufficient; for they might point to a paralysis from embolism, thrombosis, cerebral hemorrhage, lead-poisoning, etc. It is the history of the case which gives us the key to the mysteries of the affection. Then we will readily notice, by observing its general characters, that it is neither an ordinary hemiplegia nor a paraplegia,—a hemiplegia generally being related to an organic cerebral lesion, a paraplegia being in most cases connected with spinal disease. But now let us suppose that we have a little patient before us, and that we are trying to gain the necessary information from a relative who accompanies the child. She tells us that the doctor in attendance had said, a few weeks ago, that the child had diphtheria, and on further questioning we elicit the fact that sore throat did exist, and that there was a gray appearance of the mucous membrane lining the fauces. But even if the doctor had so stated, it would not always follow that his opinion was correct, especially if diphtheria is not prevailing epidemically; for with a certain class of practitioners the prevalence of diphtheria in their practice is a source of astonishment. An ordinary sore throat, a tonsillitis, etc., generally recovers with little or no treatment; but it is quite a meritorious action to cure a diphtheria: hence they often either ignorantly or willfully exaggerate the morbid condition. This is the reason for the proneness on the part of some physicians to diagnosticate many trifling affections as cases of diphtheria. And, again, certain unscrupulous and speculative charlatans do not

hesitate to manufacture so-called cases of diphtheria : they take a piece of nitrate of silver, pass it over the pharynx, and, after this has formed a white coat, they call the mother to see what a fearful membrane is covering the child's fauces ! That such things are done is not fancy, but, I regret to say, a fact ; and such men ought to be in the penitentiary, where they properly belong. So you see that the mere fact of the statement that the child had recovered from an attack of diphtheria is not a sufficient proof of the truth of its having existed. Some persons lie through design, others make false statements through ignorance. Trousseau contends that many good diagnosticians sometimes find it difficult to decide between some forms of membranous sore throat and certain mild forms of diphtheria. The history, therefore, does not always settle the question ; but it helps to throw light on the subject. But you know that all diseases, like all men, have their peculiarities ; you never see a man without some, otherwise he would have no individuality : some are particularly pleasant, others eminently disagreeable, etc. Now, so it is in diseases : they all have their characteristics and peculiarities ; and we find at least one of these in every case of diphtheritic paralysis, which unmistakably stamps it as an affection *sui generis*. This is the remarkable, singular, and characteristic *mutability of symptoms*. For instance, one day you visit a patient affected with diphtheritic paralysis, and you find the muscles of the thigh involved ; a few days later you see him again, and you notice that the paralysis no longer affects the thigh, but has invaded the muscles of the arm, perhaps on the opposite side. Now, this will positively never happen when a paralysis is the result of a grave organic lesion, as in cerebral hemorrhage or myelitis : in these cases you may go back as often as you please, and so long as the paralytic phenomena last you will find them in the same regions and generally of the same intensity. But in diphtheritic paralysis the manifestations are changeable and fickle. Authors always ascribe great importance to this change in the symptoms, and I advise you to treasure this fact up in your memory, for this mutability, this fickleness, this vacillation as regards the seat of the paralytic phenomena is pathognomonic of diphtheritic paralysis. This fact, in connection with the history of the case, is generally sufficient to settle the question.

Before speaking of the pathology of the disease, there are two points to which I wish to refer. The *first* is, that it was formerly contended by several writers that as it is the velum palati which is generally affected, and sometimes even the only part involved, therefore the paralysis is merely an accidental circumstance, the result of inflammation of the muscular tissues, and not a true diphtheritic paralysis. They claimed that the reason is to be found in the fact that the muscles of the soft palate are the particular ones inflamed in diphtheria, and that it is in consequence of the inflammatory processes which have affected them that the impairment of the muscular contractility is produced. This is exceptionally true, and may explain some cases; but how will it account for the following circumstance? Suppose there is an epidemic prevalence of diphtheria, and that a patient has pneumonia, and some physician blisters the poor fellow's chest, only increasing the pains, with no compensatory advantage. By a blister is produced an abrasion of the cuticle, and this, during the prevalence of diphtheria, will invite the diphtheritic inflammation to the sore surface, which will become covered by a false membrane, the result of a true cutaneous diphtheria. But suppose that about one month after this *cutaneous* diphtheria the velum palati becomes affected, and at the same time a nasal intonation of the voice and a dysphagia manifest themselves: surely you cannot accuse a throat-inflammation of having in this instance produced the paralysis, for there may never before have been an inflammatory condition of the muscles involved, and there is no possible connection between the throat and a cutaneous diphtheria. The true explanation is that a toxæmia, a dyscrasia of the blood, has existed; and a patient may die of diphtheritic blood-poisoning even before a membranous exudation has commenced to cover the pharynx. This happens not only in diphtheria, but also in several other malignant diseases. I have seen many children die of convulsions the result of scarlatina-poisoning before the rash had made its appearance. Certain organisms have not the power to resist a blood-poison of a certain intensity, even when present in a small amount, and they readily succumb to the effects.

I recollect the wife of a physician who died of diphtheria before any one knew what ailed her. She had been enjoying perfect

health up to that time, and went to bed in the usual state; suddenly, during the night, she awoke, and was in great distress: she was choking, could hardly breathe, and exclaimed that she was dying. And really a couple of hours afterwards she expired; and of what? Of a malignant form of diphtheria, which she had contracted while nursing a sister who had died of the same disease. Her system had become invaded and completely overwhelmed by the blood-poison before there was time for the appearance of an exudation. Vigorous children often die just as rapidly from scarlatina, and adults from cerebro-spinal meningitis, notwithstanding blisters, calomel, gelseminum, *et id omne genus*.

This brings us to the consideration of the *second* point, which is the connection between *albuminuria* and *diphtheritic paralysis*. You have heard me say that in certain cases of diphtheria albuminuria is present; and it has been contended by a few authors that when paralytic phenomena, together with visual disturbances, follow a case of diphtheria, they are due to the existing albuminuria, whereby they are explained. What are the peculiarities of blood-poisoning from albuminuria? We know that it is usually due to an affection of the kidneys, in consequence of which these organs no longer perform their physiological duties; the blood in the renal veins stagnates, its serum accumulates in the cellular tissues, and its albumen transudes with the urine; but the principal function, namely, the elimination of urea from the blood by the kidneys, is interfered with, resulting, consequently, in uræmic poisoning from the accumulation of excrementitious matter in the vital current. But how does this uræmic poison act? One of the principal features of uræmic poisoning is convulsions, the next is delirium, and the last is coma, which generally closes the scene. Now, when I described to you the ordinary symptoms of diphtheritic paralysis, did I speak to you of convulsions, did I mention coma, or did I say anything about delirium? In the severe or adynamic form, yes; but not in the mild form, for in this variety the symptoms are only of a paralytic character, producing either sensory or motor disturbances. You see that the paralytic phenomena in diphtheritic paralysis, and the nervous symptoms which accompany albuminuria (convulsions, delirium, and coma), are entirely distinct clinical features. That albumi-

nuria oftentimes accompanies diphtheria there is no doubt, though it is surely not present in the vast majority of cases; but that the phenomena of diphtheritic paralysis are dependent upon albuminuria is an erroneous conclusion.

These remarks bring us to the *pathology* of the disease. How are we to explain the pathology? How is it that a paralytic disease sometimes follows the introduction of the diphtheritic poison into the blood? I have so frequently spoken of the varied effects of different blood-poisons—I have so tenaciously dwelt upon the important fact that all tissues of the body, all organs, the nerve-centres especially, need a healthy normal blood, not a poisoned blood, for the proper performance of their individual physiological functions—that I feel confident that you are well prepared to understand the true nature of the disease. In the first place, the single fact of the mutability of symptoms necessarily precludes the idea of an organic disease of the nerve-centres. If such an organic disease existed, the characteristic mutability of the symptoms would not be found; the phenomena would no longer be transient or evanescent, but would be permanent. This difference is an important point in the pathology, and shows that diphtheritic paralysis is simply a *functional* derangement of those nervous centres which preside over the different muscles and parts affected.

The next question is, What causes the functional derangement? It is caused by the morbid influence exercised on the nervous centres, through the blood, by the diphtheritic poison, whatever that may be. That it is a specific poison admits of no doubt; it is as specific as the poison which produces typhus or typhoid fever; and it is its deleterious action on the nerve-centres which gives rise to the abnormal phenomena. The blood is poisoned and impure, and, as in all cases of toxæmia, it is no longer fit to supply the different organs with the necessary nutrient matter. It is according to the quantity of blood-poison in the body, and the susceptibility of the patient's system, that the morbid phenomena are grave and lasting. Perverted nutrition is the solution of the question.

The *prognosis* of the mild form of diphtheritic paralysis is favorable; still, the noxious influence exercised by the poisoned blood over the cerebral and spinal nerve-centres will be felt for a

certain length of time, and a certain period has to elapse before they recover. But these phenomena are not entirely peculiar to diphtheria: sometimes a case of smallpox is followed by paraplegia; any exanthematous disease may have the same results; and even typhoid fever occasionally gives rise to paraplegic manifestations. In each of these cases the action is the same: it is the influence of a blood-poison on the nervous centres which alters their functions for a certain length of time. So much for the prognosis. You understand that the reason why it is favorable is because the disease does not produce any organic lesion. If the blood-poison led to an inflammatory condition, or to certain organic lesions, and if the patient should die of an intercurrent affection, a post-mortem examination would necessarily reveal these structural changes; the prognosis then could not be so favorable. But this is not the case: a post-mortem examination reveals nothing, at least with our present means of observation; no traces of the particular chain of causes which led to the paralytic phenomena can be found.

As regards the *treatment*, I can sum it up in two words,—tonics and nutrition. The powers of the individual having become prostrated during the diphtheritic disease, you should sustain the vital powers by the best mode of treatment possible. Such remedies as quinine and the preparations of iron and strychnia often produce good results. Strychnia especially can be recommended, on account of the remarkable selective affinity which the nerve-centres have for this substance: as soon as it arrives near them it invites an afflux of blood to these parts. I really do not know any other remedy which will have the same beneficial effects.

Always be on the lookout for intercurrent diseases, especially of the respiratory organs, and try to prevent their accession; and where paralytic phenomena exist, you might try the different forms of electricity, being careful to use gentle currents, so as not to add another shock to the many which the patient has already sustained.

LECTURE XXXVI.

PARALYSIS AGITANS.—ESSENTIAL INFANTILE PARALYSIS.— PROGRESSIVE MYO-SCLEROSIC PARALYSIS.

Diseases confounded with Paralysis Agitans.—Tremor.—Etiology.—Two Pathological Conditions.—Symptoms.—Duration.—Festination.—Treatment.—Essential Infantile Paralysis.—Paralysis after repeated Convulsive Attacks.—Congenital Disease.—Cerebral Hemorrhage in Children.—Clinical History.—Fever.—Paralysis.—Diminution of Temperature.—Impairment of Sensibility, of Reflex Irritability, and of Electro-Muscular Contractility.—Deformities.—Prognosis.—Morbid Anatomy.—Treatment.—Progressive Myo-Sclerotic Paralysis.—Symptoms.—Pathology.

GENTLEMEN,—I shall now speak of various forms of paralysis, and the first I shall consider is the affection called *paralysis agitans*. It is a disease of the nervous system, and depends for its production upon different modes of causation. Sometimes the phenomena can be referred to an organic disease, in which cases certain pathological lesions and alterations of nerve-structures can frequently be found after death. In others, no such tissue-changes can be found; it is then classed as one of the neuroses. A neurosis is a disease without any constant or uniform anatomical characters. The fact is now established that paralysis agitans is in some cases an organic disease of the nervous system, while in others it is simply a functional nervous derangement, and then belongs to the class of the neuroses. It is an affection which occurs under many different and variable circumstances, and its nosology has often given rise to much discussion. At all events, when the paralysis is simply a disease of functional character, or a neurosis, it consists in a derangement in the normal action of the motor nerve-cells; and when the disease depends for its manifestations upon certain histological lesions or disintegrations, such as may be evident to the eye by means of the microscope, it is an organic disease of the nervous system, especially of the motor nervous centres, and generally of the pons varolii or of the medulla oblongata. On post-mortem examination you will find these

parts more or less sclerosed; this change may not be constant and uniform in all cases; but in all instances of this disease where post-mortem examinations have been fruitful of results there has been found a sclerosis of that portion of the intra-cranial prolongation of the spinal cord known as the mesocephalon, and formed by the tubercula quadrigemina, the medulla oblongata, the pons varolii, etc.

Different affections are commonly confounded with paralysis agitans: one of these is *multiple cerebral sclerosis*; another is *cerebro-spinal sclerosis*; but more frequently than all others is *senile trembling* confounded with paralysis agitans. This senile trembling is a peculiar continual tremor, common to certain persons of an advanced age, and generally confined to the hand or to the head, mostly to the latter. There is usually an incessant lateral motion, an uninterrupted nodding of the head, which is always kept up during the waking hours, and sometimes even persists during sleep. If you have ever read Watson's "Practice of Medicine," you will remember that he relates the case of an aged woman, whom he frequently met in church, who had this remarkable tremor, or "bobbing of the head." This was a case of senile trembling, and the motion was confined to the cervical muscles alone; therefore it differed from paralysis agitans, as I shall soon have occasion to explain.

Until within comparatively a very recent period, multiple cerebro-spinal sclerosis, multiple cerebral sclerosis, and paralysis agitans were inextricably confounded under one group,—that of paralysis agitans. The first two are organic diseases of the nervous system, whilst the last—paralysis agitans—is sometimes a neurosis. The researches of Charcot and Vulpian, Meredith Clymer and Hammond, have thrown great light upon the diagnosis, nosology, and pathology of these affections.

In all cases of paralysis agitans there is one peculiar, characteristic, pathognomonic symptom,—the noticeable weakening of the nervous stability. You know that in the normal state of all individuals there is a sufficient amount of nervous tonicity to maintain a uniform static condition of the muscles while they are at rest. When a person is in a state of repose, he is not using his muscular powers, and the nerve-centres controlling the action of the voluntary muscles are in a condition of recuperation. It is

when one is exhausted from protracted exertion of muscular action that he sits or lies down, and simply in order that the nervous forces may recuperate. If the muscular actions were persistently kept up, a period of exhaustion would sooner or later arrive, and the muscles would become unable to perform their ordinary duty.

Recollect a law upon which I have dwelt considerably and with great stress, that there can be no excessive physical expenditure of any kind without a corresponding exhaustion: therefore, if the muscular action has been too excessive, the exhaustion may be so profound that recuperation may be very difficult. In the disease which now engages our attention, the muscular power is constantly interfered with from deficient innervation, which, should it continue for a certain length of time, cannot fail to result in a paralysis. Hence the disease is well named paralysis agitans. The word *agitans* refers to the tremor, which is the peculiar feature during the earlier stages of the disease, to be followed ultimately by a paralysis. The paralytic phenomena, therefore, are developed only after the muscular tremor has existed for a certain length of time.

If we closely analyze this *tremor*, or shaking, we shall find that it consists of alternate contractions and relaxations of certain muscular fibres; these alternations are not evident and apparent simply on account of the great rapidity with which they are accomplished and succeed one another, and it is the rapidity of the movements that causes the peculiar appearance of the tremor. Of course, certain pauses of innervation exist during an inappreciable interval, and a slight cessation in the muscular agitation may occasionally be noticed; still, the peculiarity of the tremor is that it is constant, or, at least, would appear so to any casual observer.

To reiterate: the first phenomenon characteristic of paralysis agitans is the tremor,—the peculiar rhythmical shaking of the muscles,—which, as the disease progresses, gives way to well-marked paralytic phenomena. When paralysis agitans occurs, it is generally in persons between fifty and sixty-five years of age, or thereabouts, being a disease which, as a rule, attacks only individuals who have passed the meridian of life. As regards the *etiology* of the affection nothing very definite is known. It is said to be caused by exposure to humidity, especially if the patient be of the rheumatic diathesis, perhaps, also, by over-indulgence

in the sexual act, and very often by sudden and violent emotions, such as anger, fright, or other sources of moral agitation. Paralysis agitans has been termed by Trousseau an inexorable disease, because of its rare cure. It is true that in a few cases, at the age of about thirty or thirty-five, cures have been wrought. In these cases, however, the disease was a neurosis; but in the vast majority of cases, when the individual is more advanced in life, there is an organic change of the constituents of the pons varolii or the medulla oblongata, etc., of a sclerotic and progressive character.

If you have carefully followed me thus far, you will have noticed the fact that paralysis agitans admits of a pathological division into *two kinds of affections*: one, a neurosis, without any uniform pathological characteristics; the other, an organic disease, a permanent lesion of the mesocephalon, or of the intra-cranial prolongation of the spinal cord. These two distinct types or varieties of paralysis agitans you must always bear in mind.

The *symptoms* of paralysis agitans are about as follows. There is a development, sooner or later, of a tremor of the voluntary muscles, generally, at first, in one extremity; the tremor may be either in the lower or in the upper extremities, and it is often aggravated or intensified under the influence of emotions, or of perturbing causes of a moral nature. When the patient is quiet, the tremor may not be very apparent. There is a tendency to the development of exacerbations, which occurs frequently when the attention of the patient is drawn to his affection, or when he speaks about it to his physician, etc. At first the tremor presents intervals, and is not constant or continuous. An aggravation is most likely to show itself when the lower extremities are the ones affected; and if the disease is well pronounced, you may sometimes distinctly hear the foot rattle against the floor with a violence corresponding to the intensity of the tremor. The disease being inexorable and progressive, it gradually extends from one part of the body to another,—generally from below upwards; sometimes one leg is first attacked, and soon the upper extremity on the corresponding side becomes affected; or, again, the other leg may be implicated, and the tremor travel upwards. There is always a methodical or systematic progression, the tremor never attacking a limb at random or without a certain order of succession.

At first, then, the tremor is not developed without a perturbing cause, and finally all the voluntary muscles may become involved. It is subject to certain intervals, and subsides during sleep; but as the disease advances, the continuous movement is kept up even during the night; and in consequence of this persistent, unnatural condition of unceasing motion, the patient loses sleep and becomes very much debilitated. This debility is a serious matter, for it predisposes to excoriation and sloughings, which not infrequently result from the violent convulsive movements. As regards the course of the disease, it has been said that paralysis agitans may last seven, eight, or ten years, and even longer; but there is no definite limitation so far as time is concerned; the patient may not die of it at all, but may be carried off by an intercurrent disease which will not have the slightest connection with the paralysis agitans.

There is in this disease a remarkable symptom, known as *festination*. This exists in consequence of the fact that when the patient walks there is a tendency to throw the body forward: he would inevitably fall if he did not walk very rapidly, and often run, in order to maintain his equilibrium. This peculiarity of gait has caused a French author to remark that "the patient appears to be running after his centre of gravity." Therefore these people can better walk fast than slow, and can quite easily ascend with rapidity a flight of stairs, while it would be impossible for them to come down unsupported. There is also a tendency to approximate the elbows to the thorax, and the arms are generally held in an oblique position. As the disease progresses, the difficulty in walking increases, the patient soon finding it impossible to advance unless some one walks backwards in front of him, allowing him to place his hands upon his shoulders for support. I suppose you all now understand what is meant by festination. I should add one remark regarding it, which is, that it may exist as a symptom in a few other affections, such as multiple cerebral sclerosis, first described by Dr. Hammond, and multiple cerebro-spinal sclerosis. The tremor which was at first only partial now becomes general, regular, and rhythmical in character, almost like a cadence, so that you might exactly time the patient as regards the number of the shocks. The convulsive agitations finally lead to certain paralytic phenomena, and

these become more marked as the disease progresses. The increase of the paralysis can readily be ascertained by means of the dynamometer; and you find that every day the muscular efforts diminish in power, till at last they no longer manifest themselves. The sphincters also become affected, and the saliva continually runs out of the mouth. These advanced cases are really sad to witness, and well merit sympathy.

So far as the *diagnosis* is concerned, it is hardly possible to make a mistake, if you bear in mind that the tremor is always the initial phenomenon, being in all cases followed by the paralysis. There are, as I have already stated, certain forms of sclerosis in which the paralysis precedes the tremor; but in *paralysis agitans* this is not the case, the tremor appearing first: it is continuous, and has a tendency to become general and involve all the voluntary muscles of the body. You should also consider that the tremor in *paralysis agitans* is not dependent upon any particular toxic influence, such as a mercurial or alcoholic poisoning, or any pathological condition of the blood. These should always be excluded before making the diagnosis.

As regards the *treatment*, there is a remarkable cure on record by Elliotson, of England. He overcame the disease by the free use of carbonate of iron; and since then this remedy has often been tried, but always without success. Romberg makes use of the cold douche and the warm bath; Benedikt, of Vienna, and Jaccoud and Remak, of Paris, recommend the systematic application of the continuous current. All these methods, however, are of little avail. The worthlessness of treatment is probably the most striking feature connected with the disease. Still, the fact that you cannot conquer a disease by therapeutic measures is no reason why you should not be able to diagnosticate it. As intelligent practitioners, you should understand the characteristics of all diseases which you may be called upon to treat, and then, should you come in contact with other well-educated physicians, you will not be forced to expose an ill-becoming ignorance.

ESSENTIAL INFANTILE PARALYSIS.

The form of disease we shall next consider is one that may often attract your attention. Familiar to all practitioners, it has the merit of being sometimes amenable to treatment, especially if

taken in time. I mean *organic* or *essential infantile paralysis*: organic, on account of the structural lesions which it presents in the spinal cord; infantile, because it attacks only very young children. The disease is not one of reflex origin; it is not a reflex paralysis, nor a mere functional derangement of certain nerve-centres, but consists in destructive changes of a peculiar character, and is therefore called essential infantile paralysis. The first symptoms of the disease are vague and indefinite as regards their manner of development; but the fact is that there are two principal varieties of infantile paralysis, just as there are two classes of paralysis agitans. One form occurs in some children after certain very evident brain-symptoms, and a hemiplegia, or other paralytic phenomenon, is often directly traceable to cerebral troubles. This may occur *after repeated convulsive attacks*. You may observe that when a child is suffering from eclamptic attacks one side of the body is more affected than the other, especially if the convulsions are of frequent occurrence; so that if there is a paralysis on one side of the body, following repeated eclamptic attacks, and if the paralysis is on the side on which the convulsive phenomena were most distinct and evident, in nine cases out of ten you will be correct in your suspicion that an organic disease of the brain is the immediate cause of the paralysis.

In some cases the disease is *congenital*, and the paralysis is manifested so early that it is undoubtedly the consequence of an original defect of conformation. It may be well to note in this connection that some physicians contend that children are never subject to *cerebral hemorrhage*. I take the opposite position, though admitting at the same time that cerebral hemorrhage in young children is a pathological condition of extremely rare occurrence. As argumentative proof of this point, I refer to Dr. J. Lewis Smith, of New York, who reports in his work several cases of cerebral hemorrhage in children, followed by a permanent hemiplegia. My reason for laying stress upon this matter is, that if you read West you will find that whilst he admits that cerebral hemorrhage may occur in children, he does not regard the paralytic symptoms so seriously. He does not consider them of very little importance, but as a general thing he refers to them as being much less grave as compared with the same condition in

adults. In children, paralysis is obstinate and often permanent in its effects, but is not usually a cause for great apprehension so far as imminent danger is concerned. When paralytic symptoms are developed in an adult there is always cause for great alarm ; and well there may be, for as a general thing paralysis is a very serious matter. In fact, paralytic manifestations of any kind portend, very frequently, organic cerebral disturbance. I agree with Dr. West thus far, that paralysis in children is trivial as regards the imminent danger to life, but not at all as regards the statement that it is easily amenable to therapeutic measures ; for paralysis in children is a symptom which I have always found it very difficult to overcome. A paralysis from *spinal* troubles is more frequent in young children, and is also very difficult to relieve. It is a misfortune, as Dr. West says, that in these cases it is almost impossible, on account of their long duration, not to lose sight of them. They are generally taken from one physician to another, rendering it very difficult to follow the subsequent history or to know what has become of them. I wish you to bear in mind that when a paralysis follows eclamptic attacks, especially if these attacks have been of frequent occurrence, and if, moreover, the paralysis is on that side of the body which was most severely convulsed, the probability is that it is dependent upon a brain-lesion, or that it is symptomatic of a cerebral disturbance of some kind. A practical point which I may mention is the great importance of closely examining a child after a fit. A paralysis, especially if only slight, is sometimes easily overlooked, the children being so small and young—generally from six months to about three years of age—that they are seldom able to walk, and hence the paralysis may escape notice. So it is a matter of importance to ascertain after a fit if any paralytic phenomena are present ; for should they exist, you might, by judicious means, prevent an atrophy of muscles, which would be inevitable were the paralysis not recognized in time.

The ordinary history of cases of infantile paralysis is as follows. Infantile paralysis generally succeeds a marked organic tissue-alteration, and is usually preceded by cerebral or spinal symptoms. Still, it very frequently happens that there is nothing whatever in the history or symptoms pointing either to the brain or to the spinal cord as the source of the disorder. A child may

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sit down on a cold door-step, go to bed in good health, and the next morning on awaking a paralysis may affect one of its limbs. This may be all the history; and yet you may have a very difficult case of paralysis to deal with. These are by no means rare and exceptional cases, for they happen frequently, and may greatly annoy you in practice. The paralysis may follow troublesome dentition, and in reality it generally occurs about the time that children are undergoing the changes in the organism which accompany dentition. It may likewise follow some eccentric irritation, as that of intestinal worms. All that you may sometimes be able to learn about the precursory phenomena is that the child was unwell, had a slight fever, but nothing of sufficient importance to prompt the mother even to send for a physician. The disease often follows some of the exanthematous affections. The *fever* then is all that may precede the paralysis, and it may also happen that you first see the child after the lapse of a considerable time from the commencement of the paralysis; the parents may not have observed it, and you may overlook it yourselves unless you are very careful in your examination, for frequently, in very young children, the early symptoms of paralysis are not particularly well marked and evident.

When a child becomes paralyzed, you may notice a marked *diminution of the temperature* in the palsied limb; the skin will be bluish or livid, and will be cold to the touch. This is more evident in children than in adults; the temperature may be as much as seven degrees below the normal standard. Again, the paralysis is not always complete, and the child may be able to use the paralyzed limb to a limited extent. There is no rule as regards the intensity of the paralysis, or its particular location in certain regions: there may be a hemiplegia or a paraplegia, or a paraplegia *plus* a paralysis of one upper extremity; or there may be a limited number of voluntary muscles involved; so that the paralysis could not be classed under either of the above divisions. The *sensibility* of the paralyzed limb is but little impaired, and the *reflex irritability* may be totally abolished. There is also a tendency to rapid atrophy of the muscles involved, which atrophy is not at all in proportion to the completeness of the paralysis. This rapid degeneration of muscular tissue, with corresponding fatty substitution, is often so marked as to be very

evident even a short time after the commencement of the paralysis, on comparing the affected with the healthy limb. This rapid atrophy is almost pathognomonic of this disease. The *electro-muscular contractility* is always impaired, and is sometimes even entirely lost, there being no response whatever to either the faradic or the continuous galvanic current. Some of you may imagine that you will not have to treat any such disease, supposing that all these paralytic affections will be sent to specialists; but let me undeceive you. These cases belong properly to the family practitioner; and if he cannot cure them it at least behooves him to be able to recognize them whenever they occur. I must once more remind you of the fact that the tendency to atrophy, which exists in the muscles of the paralyzed parts, is a matter of great therapeutic significance and importance.

One more point in connection with essential infantile paralysis is the tendency to the production of certain *distortions and deformities*: these, sometimes, are of more interest to the orthopedist than to the practicing physician. They are partially the result of the preponderance of action of one set of unaffected muscles, which is not antagonized by the paralyzed ones. You are all aware that for the proper and regular performance of any muscular action there must be a simultaneous relaxation and contraction of two antagonistic sets of muscles. For instance, the flexors antagonize the extensors, and when the flexor muscles contract with a certain force there is a corresponding relaxation of the extensor muscles with the movement of the flexors; but if the flexors are paralyzed, either partially or completely, the preponderance of action of the extensor muscles will necessarily draw the limb to one side, and produce a deformity which may become permanent and require the assistance of the surgeon for its correction. In this way originate many of the cases of club-foot which are sent to the orthopedic specialist for relief.

The prognosis of essential infantile paralysis is in strict relation with the retention of a certain amount of electro-muscular contractility in the affected muscles. Should there be no response to any form of electricity, be it the faradic or the interrupted galvanic current, it will be useless to undertake the relief of the little patient by any therapeutic measure. But should there be even a very limited response, you will be perfectly justified in

attempting a cure, which, after a certain length of time and with perseverance, you may succeed in accomplishing. Such cases are very tedious, and will require, in addition to skill, an immense amount of patience on your part. Never commence treatment unless a certain amount of muscular contractility be left, for upon this depends the prospect for the cure.

As regards the *morbid anatomy*, we divide the pathological conditions into three classes, as affecting the muscles, the nerves, and the spinal cord. Without entering into a long disquisition in reference to the pathology of essential infantile paralysis, I shall simply state that, with our present means of knowledge, we expect to find in these cases, generally, a certain degenerative tissue-change in the ganglionic nerve-cells of the anterior cornua of the spinal cord, of an atrophic character. The changes in the nerves are a shrinking, an atrophy, of the anterior roots of the peripheral nerves which supply the muscles affected; and the change in the muscles consists in a marked tendency to the production of an atrophic condition,—not only an atrophy, but also a muscular wasting, accompanied by a fatty substitution in the ultimate muscular fasciculi; and this is always characteristic.

As to *treatment*, there is only one remedy which I hold in high estimation, which consists of the preparations of *nux vomica*. Should you be inclined towards the alkaloids,—strychnia, brucia, or their salts,—you may use them, but at the same time carefully and constantly watching your patient: with the child continually under your observation, you will be prepared to stop the administration of the dangerous remedy at the first note of alarm. My advice to you is to use *nux vomica*, in the form either of tincture or of an extract. The chalybeate preparations, also, as well as quinine, are often of immense advantage in toning up the nerves and helping them to perform their physiological duty; but the treatment of the paralyzed limbs which is most important in simple cases consists in friction, shampooing, the cold douche, and stimulating liniments along the spine. Should these means prove unsuccessful, and a tendency to atrophy of the muscles manifest itself, the only agent in which I have any confidence—provided the disease has not gone too far—is one of the forms of electricity. The form to be used depends altogether upon the response which its administration elicits on trial. The continuous galvanic current,

slowly interrupted, produces the best results in the vast majority of cases. Should this have no effect, use the faradic current, and *vice versa*. If the muscles respond to both the faradic and the continuous currents, use the one which seems to exert the greater influence. Dr. Hammond was the first person in America to use the primary current in infantile paralysis. It may undoubtedly take a very long time to produce a recovery,—sometimes from six months to three years, and even longer; but should you be successful, or even partially so, you may well be satisfied at having attained so desirable a result, and at having arrested the deformities and prevented the helplessness to which your little patient would otherwise have been doomed.

PROGRESSIVE MYO-SCLEROSIC PARALYSIS.

There is one more form of paralysis to which I wish simply to refer, the affection being one of exceedingly rare occurrence,—so rare, in fact, that it is doubtful whether you will ever meet with a single case. This course of lectures would, nevertheless, be incomplete were I to omit to mention it. The fact that a disease is seldom met with is surely not a sufficient reason to preclude the knowledge of its characteristic features.

I shall, therefore, proceed to describe it, but shall have to rely entirely upon the books, for I have never seen a single case myself, and Dr. Hammond says that he has seen but one. This disease is called *progressive myo-sclerotic paralysis*,—a rather long name, but a very appropriate one, for it is a paralysis progressive in character and affecting the muscles. That it is a sclerosis I shall soon explain. It is a disease almost entirely of childhood (cases have occurred in adolescence and in adults), and we expect to find it only in very young children. It attacks male children principally,—rarely, or never, females.

The *symptoms* are, first, a remarkable weakness in the lower limbs; next, a peculiar waddling gait. I say waddling, because the manner of progression in these patients resembles the mode of locomotion of ducks. Authors also state that there is always a widening of base, which is apparent when the children stand still, as well as when they walk. It is this widening that greatly contributes to the peculiarity of the gait. Another important feature is the hypertrophy of the *gastrocnemii* muscles:

this is characteristic of the disease, for you will notice first an increasing enlargement of the calves of the legs, which afterwards extends to the thighs and gluteal muscles; and it is stated that the muscles of the upper extremities, as well as those of the trunk, may finally become affected.

Another symptom in these cases is *cordosis*. This is a peculiar bending of the spinal column, in which a lead suspended from the upper portion of the spine will not touch the sacrum, but will clear the buttocks. This is, therefore, a regular incurvature of the spine. In Hammond's work on "Diseases of the Nervous System," and also, I believe, in Aitken's work on Practice, as well as in Duchenne's classical treatise, you will find plates giving a representation of this condition.

As the disease advances, there is a gradual development of cerebral symptoms; and, so far as our art is concerned, there is no way to relieve them, the disease being progressive in character, and going on from bad to worse till the patient's death ensues.

To recapitulate the symptoms, you find, first the weakness of the lower limbs, then the widening of the base and waddling in walking, the hypertrophy of the sural muscles, the cordosis, and, finally, the cerebral symptoms.

In regard to the *pathology*, there is but little to say. The name of the disease implies the existence of muscular sclerosis. It is, therefore, simply an undue proliferation of the muscular connective-tissue cells, which increase at the expense of the true muscular constituents. Exactly in the same manner as in progressive locomotor ataxia, there is a sclerosis or proliferation of the nervous connective tissue, the neuroglia, and an encroachment upon the ganglionic cells, leading to their absorption and atrophy,—a true muscular atrophy, and a muscular connective-tissue hypertrophy. No nerve-lesions are to be found; yet in all probability they exist. Faradization has been recommended by Duchenne, and the primary current to the sympathetic nerve and the spine by Benedikt. Duchenne reports cases in which the progress of the disease was arrested, while it was yet in the first stage, by the use of the constant current (galvanic). A case is reported by Benedikt in which the disease was of two years' duration, and was relieved, if not cured, by galvanization.

LECTURE XXXVII.

X APHASIA.

Aphasia a Symptom.—Amnesia.—Idea of Language.—Partial Aphasia.—General Aphasia.—Permanent and Transitory Aphasia.—Complicated or Uncomplicated Aphasia.—History of Aphasia.—Dr. Gall's Views.—Professor Bouillaud's Discoveries.—Internal Speech.—External Speech.—Amnesic Aphasia.—Ataxic Aphasia.—Cases of Serious Brain-Lesions without Loss of Speech.—Dr. M. Dax's Location of the Organ of Language.—The Case of "Tan Tan."—Broca's Views.—The Part in which he locates the Idea of Speech.—Lesur's Case.—Dr. Hughlings Jackson's Researches.—The Connection between Aphasia and Embolism.—Conclusions.—The Island of Reil.—Dr. Seguin's Deductions.—Dr. Simpson's Case.—Disturbance of the Intellect.—The Organ of Language in both Hemispheres.—Function of the Corpus Striatum.—Deductions.—Treatment.

GENTLEMEN,—For the subject of to-night's lecture I have selected an affection of great importance, one which will occupy a considerable time for its proper consideration; still, it is an affection which has given rise to so much obstinate discussion as regards its pathology, it involves so many questions of more than ordinary interest, that I am confident you will patiently bear with me even if I claim your attention for a more protracted length of time than that usually occupied in the delivery of these lectures. I refer to *aphasia*. The word *aphasia* is derived from the Greek *a*, privative, and *φασις*, speech: its literal meaning, therefore, is a deprivation of speech. Other words have been employed by different authors to express the same abnormal condition: the words *alalia* and *aphemia* have been used in a similar sense; they are synonymous with the word *aphasia*. It is of much importance, before proceeding to consider the different phenomena connected with aphasia, that you should understand that this is not a distinct or individual disease. In reality, *aphasia is only a symptom*, never a disease, in this respect bearing a certain analogy to paralysis. As a symptom it invariably points to a cerebral lesion, and it may occasionally be the only existing symptom of a

pathological condition of the brain,—an important fact, which I shall fully develop as I proceed.

In those particular cases in which aphasia does not depend upon an interference with the physiological action of the muscles and parts concerned in the articulation of words there is invariably an amnesia not only of speech but also of writing, and often of gesticulation.

By *amnesia* is meant a forgetfulness; and in this case we have a forgetfulness of words, as well as of the particular arrangement of conventional characters called writing; the particular and systematic utterance of certain sounds or words, and their graphic representation, being nothing else but the expression of ideas in language.

The *idea of language* is the great attribute of man, by which one human being is enabled to communicate to another his innermost thoughts and be understood; but this faculty of language naturally requires for its expression certain physical manifestations, and these generally consist of words, of writing, and sometimes of gesticulation. Now, in aphasia not only are the different ways of expressing language affected, but the idea of language itself, the memory for words, the ability of forming words and grouping them in appropriate sentences by intellectual action, are also implicated, and sometimes nearly abolished. Aphasia being the result of a disease of the brain, there is no disease of the vocal apparatus, and the tongue, the vocal cords, and the laryngeal muscles may be in a normal condition, phonation and articulation not being in the least impaired. Hence there is a significant distinction between *aphasia* and *aphonia*, the latter referring simply to a voicelessness, an inability to utter a sound. This may be dependent upon some laryngeal disease, the result of some local trouble. The patient is no longer able to give expression to his ideas by speech, not from an inability to form the appropriate words in his mind, for this faculty may exist in all its efficiency, and nothing in the world may under ordinary circumstances prevent him from writing correctly and legibly. So you can clearly understand that by *aphonia* is meant an inability to speak, simply dependent upon a local trouble of the vocal apparatus. For instance, when an individual has, commonly speaking, caught cold, and is not able to converse above a whisper, we say he has *aphonia*, and not *aphasia*.

In aphasia the result of a deep-seated disease of the brain, the patient, whose mental and reasoning faculties may as yet not be impaired, is of course aware of his defect, and often takes great pains to express himself, which, necessarily, he finds great difficulty in doing; indeed, this difficulty sometimes amounts to a total inability, and he becomes fretful, annoyed, impatient, often gesticulating wildly and making angry demonstrations.

The aphasia may be either *partial* or *general*. It is sometimes partial in a restricted sense, so far as the forgetfulness of certain words is concerned. When the aphasia is general, the forgetfulness is complete, and not a single word can be brought to recollection.

An illustration of a *partial aphasia* is when a person desires a piece of bread and asks for his pantaloons; or he may be affected like a patient I have at present in St. Vincent's Asylum, who, when he wishes a cup of milk, asks for a cup of cow; or he may want a bowl of beef-tea, and say he desires a bowl of meat. Now, this does not consist in a mere accidental substitution of one word for another: there is an actual and positive forgetfulness of the words bread, milk, and beef-tea in these instances, these expressions having been entirely obliterated from the patient's vocabulary.

As I have already remarked, whenever the *aphasia* is *general* there is absolutely no recollection of any words whatever. But, you may say, if an individual has aphasia, if he cannot speak, why can he not give expression to his thoughts in writing? Simply because this is an affection of the memory of words, and this deficiency in the remembrance of words is the deep-rooted, fundamental, underlying symptom or disturbance of which aphasia is the manifestation. Consequently, general aphasia is always associated with *agraphia*, or an inability to write. It is evident that if a person has lost the recollection of a word to express a certain object he is as much at a loss to write it as to speak it.

Now, when a person is in a perfectly normal condition in all respects, he may qualify some of his ideas by gesticulations. For instance, if you wish to convey the idea generally expressed by the word "silence," you may succeed in doing so by placing the finger to the mouth in a characteristic manner; but we find that where there is an amnesia as regards words, it results not only in *agraphia*, but also in an inability to gesticulate properly.

Besides being partial or general, aphasia may also be *permanent* or *transitory*; that is, it may last uninterruptedly during the patient's lifetime, from the date of the first manifestation of the affection up to the time of his death, or it may be transient and evanescent, lasting only a few months, perhaps only a few days, or even a few hours. Again, aphasia may be *complicated*, or it may be simple and *uncomplicated*. Being of itself a symptom, aphasia may be the only symptomatic indication of the pathological condition to which it refers; but aphasia may be complicated with paralysis, with a hemiplegia, as is not infrequently the case. Moreover, the hemiplegia occurs in these cases most often on the right side of the body. However, I shall not anticipate by considering this fact at present, as I have to revert to it when speaking of the philosophy of this interesting subject.

We have now reached the important point of this lecture, namely, the consideration of the anatomical seat of the faculty of language. The questions which present themselves for discussion are the following: What are the causes of aphasia? Is there any definite part of the brain which may be designated as the anatomical site of language? Is there any particular portion of the cerebral mass which presides over the recollection of words and their use to represent our thoughts and ideas? We will have to proceed systematically in order to elucidate this matter, and I shall commence by giving you a *history* of the gradual development of the pathology of aphasia.

In the first place, Dr. Gall, a German physician, announced to the scientific world, at the beginning of the nineteenth century, that there was in the brain a distinct, separate, and individual organ, whose physiological functions were to preside over the formation and retention of words and language, and that this organ was located in that part of the cerebrum situated upon the posterior part of the supra-orbital plates.

In 1825, Prof. Bouillaud, of Paris, pointed out the remarkable connection existing between *loss of speech* and diseases of the *anterior cerebral lobes*,—a deduction based upon the study and observation of one hundred and three pathological cases. In all of these he found, in consequence of a serious disease of the anterior lobes of the brain, more or less aphasia, or impairment of speech, during life. Again, not entirely convinced by his obser-

vations, and in order to confirm his pathological deductions by physiological researches, he made a series of experiments upon dogs and other animals, and, out of twenty experiments, was successful in only one, all the others having succumbed to the shock of the operation. This one case was that of a dog, whose skull and anterior cerebral lobes Dr. Bouillaud perforated with a gimlet. The dog happening to survive, the professor was enabled to ascertain that the animal not only lost its intelligence, but was also unable to communicate with others as it formerly did; neither did it bark at any time following the operation. Now, it is impossible to ascertain whether or not the barking of a dog corresponds with the speech of man; but barking is, at any rate, a function which the dog makes use of to give utterance to some of his impulsive feelings, and the fact of this attribute being abolished by the experiment of Prof. Bouillaud is quite remarkable and instructive. He made the first important step in advance, and by his researches he proved that there are two categories of phenomena which are connected with the power of speech, namely, an *internal speech* and an *external speech*. By internal speech is meant the faculty of creating certain words representative of our ideas, and of recollecting these words; but in addition to this we have *external speech*, and this includes the faculty of co-ordinating the movements of articulation in such a manner that we may be able to utter the particular sounds which are the expressions of the action of the power of internal speech.

Aphasia itself is divided into two different conditions, *amnesic aphasia* and *ataxic aphasia*. To Dr. Hammond is due the credit of having originated this division. By *amnesic aphasia* we mean that abnormal state which prevents the recollection of the words which represent certain ideas, and also of the particular sounds to which we give utterance when we desire to convey our thoughts by certain expressions. *Ataxic aphasia* is that condition which prevents the expression of thoughts, not by a deficiency in the recollection of words, but by a difficulty of articulation. Suppose I wish to tell you something about the spectacles I hold in my hand, but that I have lost all recollection of the word "spectacles:" this loss of memory, being absolute, will constitute an amnesic aphasia. If, on the other hand, I know very well that this contrivance bears the name of "spectacles," but, by

a want of co-ordination of those muscles whose action is required in the articulation of the word, I find that my pronunciation is impaired or impeded, and that this prevents me from speaking the word "spectacles," then it is not an amnesia (for I have not forgotten the word, or its meaning), but it is an ataxic aphasia. Now, this is surely plain enough to avoid ambiguity. But I must come back to the history of the affection. Bouillaud's doctrine about the location of the organ of articulate language in the anterior cerebral lobes created much discussion, and was violently attacked in the Academy of Medicine, in Paris; until Bouillaud, confident in the correctness of his deductions, finally offered to wager five hundred francs,—to be given to any one who could by pathological cases prove the fallacy of his conclusions. For a long time succeeding this proposal the important subject seems to have elicited but little attention; at last, however, Velpeau claimed the money. He adduced to his support the case of the wig-maker, the one I referred to while speaking on the subject of cerebral tumors. You remember that I told you that this man was exceedingly bright and loquacious, and that, notwithstanding a large tumor had destroyed a considerable part of his brain, no cerebral symptoms of any consequence were apparent, even towards the close of his life. Velpeau claimed that prior to his death this man had given no external evidence of brain-disease, and that, nevertheless, a post-mortem examination had revealed the fact that an enormous scirrhus growth had invaded both hemispheres and entirely destroyed the anterior cerebral lobes. However, the medical profession in France did not put much faith in these assertions, and subsequent events went far to disprove them.

But before going any further I shall cite you a few instances to prove that *very serious brain-lesions* may occur and yet *the power of speech not be materially impaired*. The cases I shall now relate are described by Hammond, and I shall read them from his invaluable work on "Diseases of the Nervous System." The first is related by Dr. Harlow, of Vermont. The subject was a strong, healthy man, twenty-five years of age, who was engaged in ramming down a charge of powder in a rock to be blasted, when an explosion took place and the tamping-iron was driven through his head. In a few minutes he recovered consciousness, and was

put into a cart and carried three-quarters of a mile to his residence, where he got out and walked into the house. Two hours afterwards he was seen by Dr. Harlow. He was then quite conscious and collected in his mind, but exhausted by extensive hemorrhage from the hole in the top of his head. Blood, pus, and particles of brain continued to be discharged for several days, but by January 1, 1849, the wound was quite closed and the recovery complete. There was no pain in the head, but a queer feeling which he could not describe. As regards his mind, he was fitful and vacillating, though obstinate, as he had always been. He became very profane, never having been so before the accident. He lived until May 21, 1861, twelve years subsequent to the accident, when he died, after having had several convulsions. His cranium was obtained, and, with the bar, is now preserved in the Warren Anatomical Museum, in Boston. Dr. J. B. S. Jackson thus describes his skull: "The whole of the small wing of the sphenoid bone upon the left side is gone, with a large portion of the large wing, and a large portion of the orbital process of the frontal bone, leaving an opening in the base of the skull two inches in length, one inch in width posteriorly, and tapering gradually and irregularly to a point anteriorly. The opening extends from the sphenoidal fissure to the situation of the frontal sinus, and its centre is an inch from the median line. The optic foramen and the foramen rotundum are intact. Below the base of the skull the whole posterior portion of the superior maxillary bone is gone. The malar bone is uninjured, but it has been very perceptibly forced outward, and the external surface inclines somewhat outward from above downward. The lower jaw is also uninjured. The opening in the base, above described, is continuous with a line of old and *united* fracture that extends through the supra-orbitary ridge in the situation of the foramen, inclines towards and then from the median line, and terminates in an extensive fracture, that was caused by the bar as it came out through the top of the head. This fracture is situated in the left half of the frontal bone, but inferiorly it extends somewhat over the median line. In form it is about quadrilateral, but it measures two and a half by one and three-quarter inches. Two large pieces of bone are seen to have been detached and upraised, the upper one having been separated

at the coronal suture from the parietal bone, and being so closely united that the fracture does not show upon the outer surface. The lower piece shows the line of fracture all around. Owing to the loss of bone, two openings are left in the skull: one, that separates the two fragments, has nearly a triangular form, extends rather across the median line, and is four inches in circumference; the other, situated between the lower fragment and the left half of the frontal bone, is long and irregularly narrow, and is two and five-eighths inches in circumference. The edges of the fractured bone are smooth, and there is nowhere any new deposit."

The next case is related by Dr. Jackson: "Cast of the head of a man who was transfixed through the head by an iron gas-pipe, and who, to a very considerable extent, recovered from the accident. The patient, a healthy and intelligent man, about twenty-seven years of age, was blasting coal, when the charge exploded unexpectedly, and the pipe was driven through his head, entering at the junction of the middle and outer thirds of the right supra-orbital ridge, and emerging near the junction of the left parietal, occipital, and temporal bones. One of his fellow-miners saw him upon his hands and knees and struggling as if to rise, and, going to his assistance, he placed his knee upon his chest, supported his head with one hand, and with the other withdrew the pipe. This last projected about equally from the front and the back of the head, and much force was required for its removal. Brain escaped from the anterior opening, and coma and collapse supervened. In seven weeks he sat up, and in one more walked about. The right hand he used somewhat, but less well than the left. For about ten months after the accident his memory for some things was nearly lost, but during the next two months there was a considerable improvement. The accident happened on May 14, 1867, and in June, 1868, the patient, with the gas-pipe, was exhibited to the Massachusetts Medical Society. The man appeared to be in a good state of general health, and, though his mental powers were considerably impaired, there was nothing unusual in his expression, nor would there be noticed in a few minutes' conversation with him any marked deficiency of intellect."

Now, gentlemen, these are really remarkable cases, and, as in neither of them a real aphasia existed, you will begin to think

that Bouillaud's doctrine is rather unreliable. However, it has not been stated how much of the anterior cerebral lobe was affected,—whether it was the entire lobe that was destroyed, or only a portion. Therefore we shall consider a few more cases. A very interesting one is related by Trousseau. He tells us of a drunken cavalryman, who, on falling from his horse, violently struck the ground with the back part of his head, at the same time breaking his skull. He was taken to the hospital, and was delirious all the time up to his death, cursing and blaspheming incessantly. He experienced no difficulty whatever in his speech, and died about thirty-six hours after receiving the injury. Although the fall had been on the posterior portion of the head, still, the concussion had caused the anterior cerebral lobes of the brain to be jerked against the skull with such violence that, on a post-mortem examination, they were found to be in a pulpy condition.

Trousseau cites another case, which is also an interesting one. Two officers had fought a duel, and one of them had been shot in the head, the ball entering at the right temple and lodging in the left hemisphere. The splinters of bone were removed and the ball was extracted, the patient expressing his thanks immediately after the operation. His faculty of speech was not at all interfered with, and he died a short time afterwards from encephalitis. A post-mortem examination was made, and it was shown that both anterior cerebral lobes had been perforated.

But, after having heard me relate all these wonderful cases, you will naturally imagine that they do not prove much in favor of Bouillaud's theory, and that instead of clearing up the mystery these illustrations only add to your ignorance. You may be astonished to learn, also, that Bouillaud's doctrine survived, and was not much weakened, for in 1836 Dr. M. Dax, basing his conclusions upon a careful observation of one hundred and forty cases, went further, and located the power of language in the *left anterior lobe of the cerebrum*; and in 1863 his son, Mr. G. Dax, made another step forward, and maintained, with his father, that aphasia was always the result of a lesion of the left hemisphere, and also that the organ of language was situated in the anterior and outer part of the middle lobe. This announcement also led to a considerable discussion in the French Academy at Paris, some

of the learned members strongly attacking the doctrine of Bouillaud, which found an ardent champion in Mr. Auburtin. During the discussion, Mr. Broca stated that he had then under his charge an individual suffering from aphasia, who had been in that condition for upwards of fifteen years without any hemiplegia or other form of paralysis. He was answered by Mr. Auburtin, who said that if the patient died, and a post-mortem examination did not evince any pathological condition of the anterior lobes, he would positively reject Bouillaud's theory. Mr. Broca, on the other hand, pledged his honor and his reputation that if a lesion of the left anterior lobe were found, he would cease all opposition to Bouillaud's doctrine, and would support it as ardently as he had combated it.

The case of Mr. Broca's patient having become a historical one, I shall present its description. The patient's name was Le Borgne; he had been in the hospital for many years, and was accidentally brought under Mr. Broca's notice to be treated for phlegmonous erysipelas. Now, this man could pronounce only a very few words—one of which was "Tan." This word he used on nearly all occasions: when asked to state his name, age, etc., his answer invariably was "Tan." Moreover, if he wished to ask for any thing, whether bread, meat, water, etc., he always called for "Tan." Having been in the hospital so long, he was familiar to everybody, and, on account of the peculiarity of his language, he went by the name of "Tan Tan." But Mr. Tan Tan was aware of his deficiency of speech, and when he could not make himself understood he became exceedingly angry and impatient; and his anger always found relief in the repetition of the French oath "*Sacré nom de Dieu!*" which he had no difficulty at all in articulating: this oath, and the word "Tan," were the only words which ever escaped his lips. To his own misfortune, but to the great convenience of the pathologists, Mr. Tan Tan fell a victim to his erysipelas; and in his skull were hidden the mysteries which, by a post-mortem examination, Mr. Broca and Mr. Auburtin were so anxious to unravel. And what do you suppose they found? They found a limited and restricted disease of the left anterior lobe, just as had been pointed out by Mr. Dax. Now, you see, we are commencing to get an insight into this matter; and we shall make more progress before long.

Now Mr. Broca takes up the position which he had before assailed, and becomes a vigorous supporter of the doctrine that there is a definite organ in the brain presiding over the development of speech, and that it is situated in the left anterior lobe: he invites everybody to discuss the subject with him in the Academy, writes memoirs of a number of pathological cases, and goes one step further, stating that the faculty of language is not only situated in the left anterior lobe, but is also limited to a small portion of this lobe,—namely, to the *posterior part of the third left frontal convolution*.

You notice that we are steadily progressing in this labyrinth of scientific intricacies; and before long we shall be able to arrive at correct conclusions. At this stage Mr. Broca's views found a strong corroborative proof in the peculiar phenomena presented by two different cases, one of which came under the observation of Mr. Lesur, of France, the other attracting the notice of Dr. Hammond, of America. And, speaking of Hammond, I cannot permit the occasion to pass without reminding you of the fact that he is not only one of our most eminent authors, but that, as a neuropathologist, his name stands equally high with those of the distinguished investigators of whom you have heard me speak to-night. Indeed, as students of medicine, his name is already familiar to you; but, as Americans, you have the greatest reason to be proud of him. But we must revert to our subject.

Mr. Lesur's case was that of a child who had received severe injuries upon the head by the kick of a horse, the skull having been broken over the left frontal region, necessitating the operation of trephining. Lesur found that, by pressing the finger over the trephined region against that part of the brain, an amnesic aphasia was instantly developed; this, however, immediately disappeared upon removing the pressure: so that in this case an aphasia could be produced at pleasure. The case that was observed by Hammond was a similar one in its general characters.

But now comes Dr. Hughlings Jackson, an English pathologist, and he brings an array of pathological cases, which again shed a flood of light over our inquiries. Dr. H. Jackson, like many of his distinguished countrymen, is a very shrewd and learned observer. In studying the subject of aphasia, he found that in a great number of cases it was always complicated by a

hemiplegia of the right side of the body. As an explanation of this complication, he refers to the anatomical fact that part of the left cerebral hemisphere receives its blood from the left middle cerebral artery,—the artery of the fissure of Sylvius,—and not only does this artery furnish blood to the left anterior lobe, but also to the left corpus striatum. Dr. Jackson further states that in aphasia there is generally a right hemiplegia, on account of a lesion in the left hemisphere (the lesion being situated above the decussation of fibres), and that it is produced in consequence of a diseased condition—generally a mechanical obstruction—of the left middle cerebral artery.

Now, gentlemen, do you remember my lecture upon cerebral anæmia? Do you recollect that I told you that when anæmia of the brain resulted from embolism the embolus was generally situated on the left middle cerebral artery? Do you remember the reasons I gave for this fact,—the anatomical considerations for its explanation? Have you forgotten that I alluded to the fact that the current of the blood, as it was propelled towards the thoracic aorta, moved in the direction of the left carotid artery, and that a clot being detached from the fibrous structures of the heart was extremely liable to be carried along towards the brain on the left side? If you still bear all these points in mind, you will easily understand that an embolus is generally located in the left artery of the fissure of Sylvius, and that when this occurs an anæmic condition of the left anterior cerebral lobe, as well as of the left corpus striatum, necessarily follows. We shall have, therefore, the development of an aphasia *plus* a hemiplegia on the right side, the aphasia following the anæmic condition of the anterior cerebral lobe, the hemiplegia resulting from the anæmic state of the corpus striatum, a part of the great motor tract.

But let us see what *conclusions* are to be arrived at from all these statements; let us also see what light is to be derived from all these theoretical dissertations and pathological facts.

In the first place, comparative anatomy teaches us that the lobe of the insula, or the *island of Reil*, as it is sometimes called, is found in only two genera of animals,—in man and in the monkey. I speak of man as an animal as regards the physical properties which subject him to classification with all other moving and living beings. Now, this lobe of the insula is well

developed in man, is in a rudimentary state in the monkey, and is totally absent in other animals. This might point to a certain connection of origin between man and monkey ; and some philosophers, like Darwin and others, have even advanced the theory that the *genus homo* has for ancestry the primordial monkey. However, I have no desire to discuss the subject, but must confine myself to the island of Reil.

The next fact to be considered is the conclusion arrived at by Dr. Seguin, of New York. He derives his deductions from the study of five hundred and fifty-six tabulated cases, and he claims that the weight of evidence is decidedly against the limitation of the seat of the organ of speech in the posterior portion of the left third frontal convolution; for out of five hundred and fifty-six cases of aphasia, this particular part of the brain was damaged in only nineteen instances. Dr. Seguin is also opposed to the location of the faculty of language in the left hemisphere alone; moreover, his researches prove that aphasia is not invariably dependent entirely upon a pathological involvement of the posterior portion of the left third frontal convolution, but that it may result from disease in some other portion of the brain. For let us just consider the bearings of the negative results to be deduced from one single case in which it could be proven that the third left frontal convolution was not involved. Suppose it were mathematically demonstrated that amnesic aphasia can exist only in consequence of a pathological alteration in the structures of the posterior portion of the left third frontal convolution; suppose any number of well-authenticated cases have proven this beyond a doubt: if then we could just find *one* single, solitary case in which a well-marked amnesic aphasia existed during life, and which on post-mortem examination would reveal no diseased state whatever of the particular cerebral portion just mentioned, would such a single case not be sufficient to overthrow and completely subvert the theory of limitation? And, remember, Dr. Seguin did not base his observations on one such negative case, but on a large number, —on thirty-one different subjects. Again, there is a case related by Dr. Simpson, in which there was an extensive lesion of the third frontal convolution on the left side, and still no aphasia nor any paralytic phenomena were manifested during life. This is another negative result, which, in conjunction with Dr. Seguin's

cases, is sufficient to invalidate Mr. Broca's theory, and we have once more made an important step in advance. Now, it is an undisputed fact, admitted by all observers, that whenever an aphasia exists there is very apt to be a co-existing hemiplegia of the right side of the body. I have already alluded to this fact, and it proves that embolism of the middle cerebral artery is a common affection ; also, that an embolus is more likely to obstruct the left artery than the right one, for reasons which I have already given.

In these cases of aphasia there is always more or less *disturbance of the intellect* ; and this is sometimes so marked as to show conclusively that it is not a slight lesion, but a profound pathological change, giving rise to the impairment of the intellectual powers, to the loss of the faculty of language, and also to a tendency to apoplexy. Apoplexy does not infrequently follow aphasia, and it sometimes happens that aphasia is the first and the only premonitory symptom of cerebral apoplexy. Another thing to be considered (and this is important, in order to get nearer to the proper understanding of the subject) is, that generally the left cerebral hemisphere is developed in children (and probably before birth) more rapidly than the right hemisphere. This depends upon the fact that the left hemisphere receives a larger supply of blood than the corresponding organ, and this point has a very significant bearing upon the phenomena of aphasia and its usual relations with a right hemiplegia. I shall fully dilate upon this in a moment.

You all know by this time that embolism is a common cause of aphasia, that an embolus is usually found in the middle cerebral arteries, but that in the vast majority of instances the embolus lodges in the *left* middle cerebral artery,—the left artery of the fissure of Sylvius. I have also frequently told you on former occasions that the brain is essentially a dual organ, composed of two symmetrical hemispheres, the functions of one being identical with those of the other : therefore I believe, with Maudsley, that it seems absurd to imagine the faculty of language or of speech to be located in only *one* side of the brain, and that it is far more rational, logical, philosophical, and probable that the different nervous centres are distributed *equally on both sides*. It might be, therefore, that the organ of language is situated

in the posterior portion of the third frontal convolutions, not only in the left but also in both hemispheres. I have exemplified the duality of the brain in several of my lectures: I have told you how it is possible to have a congestion or an inflammation in certain parts of one hemisphere, with but little evidence, or few symptoms, of a cerebral pathological condition, provided the other hemisphere be healthy and perform double or compensatory duty. This power of one portion to take upon itself the work of another part is not limited to the brain: it is common to all dual organs. You all know that it is not at all uncommon to have a disease of one entire lung; respiration may still be carried on by increased functional activity of the other. All these and many other considerations allow us safely to conclude that the organ of speech is situated in both hemispheres, and not in the left one alone, as is contended by Broca and Dax.

Now, you may perhaps ask me, if this be the case, if the organ of language be situated in both hemispheres, why is it that pathological researches have proven that hemiplegia of the right side of the body is usually associated with aphasia, while hemiplegia of the left side does not show any such connection? Does not the right hemiplegia clearly point to an affection of the left lobe? This is simply due to the fact that the left hemisphere—the left brain—is more developed than the right one, and that we more frequently use it. I have already pointed out the anatomical fact that the left hemisphere receives more blood than the right. This causes a preponderance of physiological action in the left side of the brain, and, as a consequence, that side is more frequently employed. This left side being first used in life, or at least to a greater extent than the other side, we form the unconscious habit of using it; and its development increases correspondingly, first by the increased supply of blood, secondly by force of habit. We can prove this excess of physiological activity on one side by certain very familiar facts. Are not most of us right-handed? Do we not always use the right hand, eye, foot, or any portion of the body, in preference to the left? Of course we do; and as we know that this entails a greater amount of innervation to that side, and that by the decussation of fibres in the anterior pyramids the innervation of the right side originates in the cerebral batteries of the left hemisphere, we have sufficient evidence

to justify us in the belief that we act more with the left side of the brain, that we can use it more, that we think more with it, and speak more with the left anterior cerebral lobe, than with the right. Hence it is that when aphasia is complicated by hemiplegia the paralysis is usually on the right side,—the same cause giving rise to both symptoms.

You will remember that Bouillaud located the power of language in both anterior cerebral lobes, that Dax and Broca located it in a restricted portion of one lobe on the left side, and now we have explained to you, gentlemen, that it is situated in the posterior portion of the third frontal convolution, on both sides, though the left is better developed by habit and by a larger supply of blood. But suppose we have a case of aphasia, and that the left convolutions are all in perfect health, but that the disease is limited to the posterior part of the third frontal convolution on the right side: what does that show? It merely proves that the faculty of language is situated on the right side as well as on the left, and that in this case the right side was the one most developed. And how are we to account for the wonderful instance cited by Simpson, in which the entire left anterior cerebral lobe was injured and still there was no aphasia? In all probability; by the fact that the posterior portion of the third frontal convolution on the right side was sufficiently developed to prevent the aphasia.

I have still a few remarks to make concerning aphasia, for it is important that, as well-educated physicians, you should have a clear and perfect understanding of this interesting subject. In the early part of this lecture I spoke of Hammond's division of aphasia into ataxic and amnesic. I shall now consider the philosophy of these views as regards the connection between ataxic or amnesic aphasia and the co-existence or absence of hemiplegia. If you thoroughly understand the distinction between amnesic and ataxic aphasia, you are masters of the entire subject. For the foundation upon which to base our explanations we must attach great importance to two facts: the first is the discovery of Jackson, that aphasia generally follows embolism; the second, that aphasia may exist with or without hemiplegia. Now, we have established the point that the left middle cerebral artery transmits more blood than the right: consequently, the left anterior cerebral lobe, as well as the left corpus striatum, is better

supplied with the vital fluid than the right. But what is the corpus striatum? what is its *physiological function*? It is composed of white and gray fibres, being connected with the transmission of motion and the origin of motor impulse. I spoke of this ganglion in my lectures on hyperæmia as being a part of what Jaccoud calls the system of conjunction. It communicates with the pons varolii and the medulla oblongata by one of the crura cerebri, which, as you all know, diverge, some of their fibres going to the corpora striata and thalami optici, the others forming the medullary masses of the hemispheres. The matter of the corpus striatum is continuous with that of the antero-lateral tracts of the cord, and, consequently, the corpus striatum forms a part of what is generally called the motor tract. This being understood, we shall proceed. If you take a human brain and carefully separate the anterior cerebral lobe from the temporo-sphenoidal lobe, you will find, deep in the fissure of Sylvius, a small agglomeration of isolated convolutions,—the lobe of the insula or island of Reil. An important point about this lobe of the insula is that it is covered with the same cineritious substance as the surface of the convolutions of the hemisphere; it is, therefore, intimately connected with ideation, as well as with intellection and memory; for no matter in what part of the cerebrum these convolutions and its gray substance are situated, they invariably form a part of the highest and noblest attribute of man,—the faculty of ideation, intellection, or reasoning. But the next point of importance is that immediately below this gray substance we find what is called the extra-ventricular nucleus of the corpus striatum; and this is in direct relation with the motor tract and the manifestations of physical phenomena such as muscular action. Suppose, now, that we injure the outer portion of the island of Reil, that part which is composed of gray substance and in connection with the cortical portion of the hemispheres: what will be the result? We shall have an amnesic aphasia, a loss of memory, and an impairment of the intellect, the part affected being connected with the evolution of memory. But suppose we go still deeper, and injure the extra-ventricular nucleus of the corpus striatum: what then? We produce an interference with the physiological functions of the motor tract, and an abolition of the power of motion follows, manifested as a hemiplegia; but the abolition of

motion will necessarily be accompanied by an ataxic aphasia, and there will be a deprivation of the power of articulation, simply because the proper nerve-force for the co-ordination of the muscles of articulation is no longer evolved. There will be no amnesic aphasia unless the gray substance on the surface of the lobe of the insula be also injured. For instance, here is a hat: if, now, this gray matter of the island of Reil be affected in my brain, I shall still be able to speak, but I shall have entirely forgotten that this particular object is called a hat; and, of course, if I have forgotten the word I shall be unable to pronounce it; but if my corpus striatum be injured, then the muscles of articulation, being no longer co-ordinated, will not allow me to give expression to my thoughts by language, though I may very well remember the word for that object. If, furthermore, both of these parts be injured simultaneously, it is evident that not only will the power of articulation be impaired, but there will be a loss of memory at the same time, or an ataxic aphasia *plus* an amnesic aphasia.

Our *deductions* must be as follows: the anatomical site of the organ of language is in a restricted portion of the brain, in the posterior part of the third frontal convolution; but, the brain being a dual organ, the faculty of language is located in the same part on either side. We have an amnesic aphasia whenever this part or the gray substance of the island of Reil is affected; we have an ataxic aphasia whenever there is an involvement of the extra-ventricular nucleus of the corpus striatum, or of any other part of the motor tract of the brain. In the one case there is a loss of recollection of words, in the other a deficiency in the power of articulation. When the loss of memory for words co-exists with an impairment of motility, then we have an amnesic and an ataxic aphasia.

I hope you are now fully conversant and familiar with the subject of aphasia; but I cannot conclude this lecture without expressing my admiration for Dr. Hammond, whose scientific investigations have thrown so much light upon its pathology.

As regards the *treatment*, aphasia being only a symptom, you can only treat the disease on which it depends; for, as I told you before, aphasia may be the solitary symptom following a cerebral hemorrhage, an inflammatory condition, or an anæmia. If the aphasia be restricted, and not complicated by a hemiplegia, a slight

blood-letting may be of some use. Trousseau cites the case of a person suffering from aphasia, where a leech to the anus was sufficient to relieve the symptom ; indeed, the aphasia disappeared while the leech was drawing ; but it must be remarked that this was but a transient condition. But whenever the aphasia is complicated by a hemiplegia, local or general abstraction of blood is of no avail, the case being perfectly hopeless in character. In aphasia, as in all other obscure brain-diseases, there is sometimes a remarkable connection with the presence of a syphilitic blood-poison ; and in aphasia, as well as in cerebral tumors, progressive locomotor ataxia, or an affection of the nervous system of any kind whatever, if there be the slightest evidence of a syphilitic diathesis, resort to antisyphilitic modes of treatment, and give mercury and iodide of potassium. From what I have repeatedly said during this entire course of lectures of the treatment of nervous affections, you know how earnestly I deprecate the use of mercury, in any of its forms ; but I must state that whenever you have a case of syphilis to deal with, I consider it to be the only remedy worth administering, either in the form of bichloride of mercury or combined with iodide of potassium.

This brings us to the conclusion of the subject of aphasia ; and, gentlemen, if my efforts have been of any assistance in clearing up this difficult question to your minds, I shall be perfectly satisfied, and feel amply compensated for the trouble I have taken.

LECTURE XXXVIII.

SYPHILITIC NERVOUS AFFECTIONS.

Nervous Symptoms in Syphilitic Patients not always dependent upon Syphilis.—Syphilis often overlooked.—History.—Period at which Nervous Symptoms appear —Nervous Symptoms in the Primary Stage.—Nervous Symptoms in Secondary Syphilis.—Nervous Symptoms in Tertiary Syphilis.—Destruction of Nervous Structures.—Pressure upon, and Irritation of, Nerve-Centres and Nerves.—Symptoms.—The Symptoms are not distinctive of Syphilis. — Diagnosis. — Abortions. — Peculiarities of Teeth.—Pathology.—Prognosis.—Treatment.

GENTLEMEN,—Throughout this course of lectures you have heard me refer to certain vices of constitution, certain cachexias, which play an important part in the causation of many different forms of nervous disease. The tubercular, the strumous, the cancerous, the rheumatic, the gouty, and the syphilitic diatheses, each and all involve the nervous system, and at times expend upon it their peculiar morbid influence. When treating of the different forms of insanity and of paralysis, as well as when considering aphasia, epilepsy, locomotor ataxia, sclerosis of the cerebro-spinal axis, and cerebral and spinal tumors, I told you that these constitutional conditions, and, above all, the syphilitic, were to be suspected and carefully taken into consideration in making your diagnosis of special, limited lesions of the nervous apparatus. I now purpose to group all these affections together, so far as the presence of syphilis is concerned. This is all the more necessary because it is a constitutional condition often overlooked by the physician, and most carefully concealed by the patient, even when its presence is suspected. In some of these cases the effects of specific medication may prove to be the only trustworthy means of arriving at a certain, clear, and incontrovertible diagnosis; in other words, specific treatment is the crucial test for the existence of syphilis.

It will be well for you to remember that people who are infected with this terrible disease may have nervous symptoms dependent upon other causes for their origin: this is especially true of those

who have arrived at that period of life when we expect to find morbid states arising from the degenerative changes which naturally appear near the close of "life's fitful fever." Another state of affairs will illustrate my meaning. Should hemiplegia and aphasia appear during the course of chronic disease of the heart or lungs, evidently caused by the detachment of a portion of a fibrinous vegetation, or of disintegrated pulmonary tissue, you would not be justified in attributing the loss of function to syphilis, although your patient evinced that disease in the secondary, or even in the tertiary, stage. As has been well remarked, "Syphilis is the scape-goat of pathology:" therefore be cautious, be circumspect, and, above all, seek to obtain *all* the evidence in a given case before you venture an opinion. This principle should always guide you in the grand science of diagnosis; but it is most necessary in the study of diseases of the nervous system, many of which are obscure enough even when examined by all the lights of modern science.

Syphilis is so common, so diffused among all ranks and degrees of people, so often present and not known, not even suspected, by the unfortunate victims,—the wives and children of the original evil-doers,—that in every doubtful case of disease, whether of the tegumentary, osseous, vascular, or nervous system, you should most carefully and persistently look for it, and be on your guard against deception. I cannot too strongly impress this important fact upon your minds; and I trust that the remembrance of my words will redound not only to the benefit of your patients, but also to your own professional reputation, and thus to the honor and glory of your *alma mater*.

In the early history of venereal diseases, especially during the fearful epidemic of the sixteenth century, all forms and degrees of nervous affection were attributed to the influence of the poison of syphilis. Then followed a season of reaction, so to speak, when this mode of causation was entirely overlooked. Within the last thirty years, the period marked by such marvelous advances in all branches of science, and especially in that of medicine, renewed investigation has shown that the nervous system, as well as all other systems of the human organism, is subject to the inroads of its destructive processes. The date of the recognition of the relationship of syphilis to the causation of insanity is still

later. Hence the necessity of your fully understanding all the bearings of the subject, if you hope to keep in the front rank of the profession.

Your professor of Surgery has already described syphilis, and has shown you illustrative cases at St. John's Hospital and the city hospital, exhibiting the protean forms which it assumes. You have seen it in all stages of development, from the initial lesion and the multiform affections of the skin and mucous membrane characterizing the secondary stage, to that final wreck of poor humanity, where the deeper tissues, the cartilaginous, the periosteal, and the osseous, are involved or destroyed in the final, tertiary period, or rather the sequelæ, of constitutional syphilis. There may be nervous symptoms developed in each of these stages of the disease, and, like all other manifestations of this direful malady, they will be grave in direct proportion to the advancement of the disease in its stages of evolution. You must remember that the period at which it has arrived is not to be estimated by the time that has elapsed since the appearance of the primary sore, but by the symptomatic phenomena which it presents upon examination. The evolution of the secondary or tertiary symptoms may be interfered with, or delayed, by the administration of mercurials; or, from peculiarity of the constitution of the individual, they may appear earlier than we would expect from an examination of the natural history of the affection, as presented by the statistics of a large number of cases; or, again, individual idiosyncrasy may exert such an influence that the usual manifestations of the secondary period may be very slightly marked or altogether absent. It is in the tertiary period, so called, that the gravest lesions of the nervous apparatus appear; and it is especially in those cases where the ordinary secondary manifestations were wanting that we are to expect these complications in the third stage.

The nervous symptoms which appear in connection with the primary stage, the period of incubation, are not prominent: they are limited to fleeting pains in the course of different nerves, headache, and some dyspeptic symptoms. These phenomena evidently depend upon anæmia, and are not at all distinctive of specific poisoning.

The evidences of the implication of the nervous system in the second stage of syphilis are more marked than in the period of

invasion. They are similar to the ordinary neuroses, or to those produced by simple inflammations of the nerve-centres and their coverings. These are neuralgias of all degrees of intensity and of divers location,—dyspepsia of nervous origin, palpitation of the heart, and meningitis, cerebral, spinal, or cerebro-spinal in character. The mental symptoms are melancholia, often of a suicidal tendency, a form of hypochondriasis, sometimes termed syphilophobia, wherein the patient can scarcely think or speak about anything but syphilis, and a lassitude or inertia of mind, called by the Germans “paralysis of energy.”

Many of these symptoms are evidently dependent upon the concomitant anæmia, and are relieved as the blood is improved; others are caused by the presence of the poison of syphilis in the circulating fluid, irritating the delicate nervous structures. There is not only “a prayer for more blood” on the part of the suffering ganglionic cells, but also a supplication for healthy blood to enable them to perform their functions; and, if they do not receive it, they soon break out in open rebellion.

In the third stage of syphilis, in addition to the osteocopic (bone-shattering) pains referable to periostitis, there are numberless shades and varieties of nervous affection,—numberless as regards their manifestations, but susceptible of classification under two grand divisions. *First*, those produced by a peculiar form of inflammation or degeneration (found only in syphilis) of the nerve-centres themselves, the ganglionic cells, connective tissue (neuroglia), blood-vessels, and lymphatic sheaths (perivascular canals) being more or less destroyed. *Secondly*, those occasioned by pressure upon the nerve-centres or nerve-trunks by the products of this same form of inflammation located in adjacent structures. These deposits may be in the meninges of the brain or spinal cord, in the bones (exostosis or caries), in the muscles, in the connective tissue, or in the sheath (neurilemma) of the nerves in any part of their course.

According to the degree of pressure upon nerve-structure will there be irritation of the tissues, producing apparent exaggeration of function where it is inconsiderable in degree, or abolition of the functions of the parts involved when it becomes excessive. Thus, hyperæsthesia, neuralgia, and epileptiform convulsions are evidences of irritation of the centres for sensation and motion,

or neuralgia of sensory nerves, and maniacal excitement when the ideational and emotional centres are subjected to irritation. When there is destruction of nerve-centres, or excessive pressure upon them, or upon nerve-trunks, we find anæsthesia or paralysis of muscles, which will be limited or general in extent according to the location of the morbid product. When the supreme cerebral centres, the ganglionic cells of the convolutions, are subjected to these processes, there are psychical symptoms which indicate their implication in the disease. There is, at first, when the destruction of gray matter has not advanced far, a mental condition undistinguishable from that found in general paresis. There are the same ideas of personal grandeur, of individual power, the same affections of articulation, and the same difficulty of locomotion. When this morbid process is far advanced, especially if epileptiform attacks have been prominently developed, a condition of complete dementia supervenes.

When I directed your attention to the different forms of insanity and of paralysis,—hemiplegia, paraplegia, Bell's palsy, etc.,—to aphasia, cerebral and spinal tumors, neuralgia, sclerosis of the cerebro-spinal axis, and locomotor ataxia, I described the symptoms of each affection, and told you that each might have syphilis as a cause. Thrombosis of the cerebral arteries, caused by a syphilitic degeneration of their coats, may also produce partial softening of the brain, operating in a manner precisely similar to *endarteritis deformans*. Single nerves arising from any portion of the cranial or spinal region may have their function interfered with or entirely destroyed. According to Broadbent and other good authorities upon the subject, the *third* and *sixth* cranial nerves are those most often affected, the inter-peduncular space being one of the most common locations of intra-cranial deposit of syphilitic inflammation.

The symptoms of these different affections, depending upon the portion of the nervous system involved, not upon the cause of the lesion, are in no way distinctive of syphilis. It makes no difference whether a nerve-centre or a nerve is destroyed by the actual cautery, by excision, by persistent anæmia or abscess, by a carcinomatous growth, by an aneurism, or by inflammation, simple or syphilitic, the function of the part will be destroyed; and if we are confined to the fact of its destruction, and the consequent

abolition of function, we are unable to form any opinion whatever regarding the causation; we are unable to arrive at any diagnosis. But, although the symptoms, of themselves, give us no information, there is a peculiar grouping of the morbid manifestations, which, according to Dr. Buzzard,* is almost pathognomonic. This distinctive characteristic is, that there are almost invariably *two or more different* affections of the nervous apparatus associated together in the same case at the same time. These "simultaneous lesions in distant parts of the cerebro-spinal axis" may thus present themselves: there may be aphasia, with or without hemiplegia, and at the same time a paralysis of a muscle, or of a group of muscles, upon the other side of the body; there may be optic neuritis with paraplegia; or there may be ptosis, insanity, or epilepsy, with paralysis of one arm or one leg. It is this tendency to localization of lesion, and at the same time to *multiplicity* of lesions, which is the most distinctive characteristic of syphilis.

When you can ascertain the fact that paralysis of single cranial or spinal nerves is produced by pressure upon the nerve after its origin and before it leaves the bony cavity, you may feel very confident that syphilis is the cause; for, as I stated in my lecture upon tumors, it is very rare to find neoplastic formations arising from the membranes. If it is a partial paralysis of the third or of the third and sixth cranial nerves, the diagnosis is strengthened. The fourth and seventh are not so often affected in this manner, although the *portio dura* may be affected by caries of the petrous portion of the temporal bone, of syphilitic origin. This was well exemplified in a case at the city hospital, which many of you doubtless remember. It was a typical case of *double Bell's palsy*, occurring in a prostitute aged nineteen years. She had had syphilis about three years. In addition to iritis and nodes upon the tibiæ and the clavicles, there was a profuse purulent discharge from both ears. Hearing was entirely destroyed, and there was complete paralysis of all the muscles of expression; the lips were widely separated, and the eyes open and staring, except when the hands were brought into requisition to close them. The countenance was as blank and devoid of all vivacity or expression as that of a cadaver. There was dribbling of saliva, and defective

* *Clinical Aspects of Syphilitic Nervous Affections*, American edition, p. 5.

articulation ; and, altogether, her condition was deplorable in the extreme. The paralyzed muscles did not respond in the least to faradization, so that there could be no doubt that there was pressure exerted upon the nerve-trunk, or that some portion of it had been destroyed after leaving its point of origin : in other words, the trouble was *peripheral*, not central.

Electrization is an important means of diagnosis. By it we are enabled to determine whether a given case of paralysis depends upon destruction of that portion of the central nervous system from which the nerve arises, and hence is hopeless ; or, on the other hand, whether it depends upon pressure from an inflammatory product which may, possibly, be absorbed. If the muscles respond to the induced current, there is central lesion ; if they do not, but react normally, or even in an exaggerated manner, to the slowly-interrupted (direct) current, it is certain that there is an interference with the conduction of nervous influence at some point between the origin of the nerve and its distribution.

Of course the history of the case, as in all forms of disease, is of the utmost importance in the formation of a diagnosis. As I warned you, this may not be attainable ; or, even if it is given in good faith, the presence of syphilis may not be suspected by the patient. In women, a history of many abortions occurring in succession should sound the note of alarm. You will immediately connect these miscarriages with the fatty degeneration of the placenta, of which your professor of Midwifery has told you. Its causation by syphilis is beyond question. In cases of epileptiform convulsions in children under seven years of age, you will examine for the wedge-shaped, deeply-notched incisor teeth peculiar to the unfortunate sufferers from *congenital* syphilis. Once upon your guard, there will be little difficulty in gaining corroborative evidence of the correctness of your diagnosis. In some cases of paralysis of cranial nerves, an ophthalmoscopic examination of the retina will disclose the peculiar changes in the retinal circulation due to the pressure exerted by an intra-cranial tumor : double optic neuritis, as Hughlings Jackson has pointed out, is the most distinctive lesion.

In epilepsy of syphilitic origin, we are often to be guided to a correct conclusion by the fact that the so-called idiopathic variety appears, in the vast majority of cases, in early life,—at or about

the period of puberty. When spasms, with unconsciousness, make their first appearance *late in life*, we are immediately to suspect a specific poisoning. If the convulsions are markedly unilateral (hemispasm), this probability is strengthened. Hughlings Jackson, who, I may here remark, has led the way in the investigation of syphilitic diseases of the brain and its arteries, as well as in many other pathological investigations, has demonstrated that this hemispasm depends upon disease of convolutions in the hemisphere opposite the side affected, and that this lesion is situated, in every case, in close proximity to the Sylvian fissure.

In hemiplegia from syphilitic thrombosis, the cutting off of the blood-supply is generally more extensive than in ordinary embolism. This form of thrombosis is met with at any age of adult life, and thus should be compared with embolism rather than with ordinary thrombosis, which occurs only in advanced life.

Paraplegia from syphilis is generally due to myelitis, often initiated by the pressure of spinal tumors. Many of you have seen the case at the city hospital of the man in ward 49, who shows some of the symptoms of locomotor ataxia, and at the same time has paralysis of the bladder and rectum, and progressively increasing loss of sensation and motion in the lower extremities. His is a history, clearly developed, of syphilis and its sequelæ. You have, some of you, noticed the nodes over the frontal bone, and also over the sacrum.

In the earlier stages of syphilis, as I have stated, there may be no post-mortem appearances to account for the nervous troubles observed during life. A little later there are developed simply the characteristic products of ordinary inflammation. There may be merely a hyperplasia, an overgrowth of the normal tissue-elements of the part affected. In the membranes of the brain and spinal cord, as well as in the neurilemma, there will be found thickening, opacity, and a deposit of lymph, which may go on to the formation of pus. Later, in the so-called tertiary period, appear the peculiar gummy deposits characteristic of that stage of the disease. This gummy material is alike wherever found: in this it differs from the product of ordinary inflammation. As Virchow observes, it has a singular frailty of constitution, a tendency to break down and be destroyed by a process of fatty degeneration. With this is conjoined localization of lesion; that

is, the whole of an organ is seldom affected at once, but the gummy deposits exist in scattered nodules in different parts of the same organ, or in many organs or structures of the body, at the same time. There is also a marked tendency to recurrence. Thus, if one formation is removed by absorption, under the influence of proper medication, or is discharged by ulceration or the formation of abscess, it is almost sure to return again and again. This gummatous degeneration may affect the neuroglia, the meninges, the periosteum, the bones, the walls of the blood-vessels, or their perivascular lymphatic sheaths. When the walls of the cerebral blood-vessels are affected, they are more or less closed by a thickening of the coats by gummy deposit disposed concentrically, in a manner precisely similar to that observed in the syphilitic degeneration of the placenta. In relation to the appearances observed in syphilitic meningitis, I cannot do better than to quote from Dr. Moxon (Guy's Hospital Reports). He says, "Syphilis attacks the surface of the brain and its membranes; it attacks them in limited spots, and spreads slowly. The morbid changes are, on the one hand, adhesions of the membranes to each other and to the surface of the brain by means of an adventitious material of firm consistence and yellow color, which may be called lymph, but is harder, tougher, and more opaque." This exudation presses upon, and interferes with, the physiological action of the gray matter and of the nerves while still within the cranium. On the other hand, it may form firm, fleshy nodules upon the surface of the convolutions at the base of the brain or within the hemispheres, generally multiple, and interfering with nervous function in the same manner as tumors having other histological elements.

Dr. J. Batty Tuke, in the "Journal of Mental Science" for October, 1874, makes an elaborate report of a case of syphilitic disease of the nervous system, in which the pathology of these affections is exhaustively treated. As the case presents many, if not most, of the characteristics noted in this form of nervous affection, and as, besides, a pathological process was developed which is almost, if not quite, unique, I shall quote it in full:

"B. A., a male, first came under observation early in 1873. His age was then fifty-two years. There was not the slightest history of any hereditary predisposition to nervous affections, nor

of any moral or physical cause except syphilis. At the age of forty-six he contracted an infecting chancre, which was followed by the usual secondary symptoms, during the persistence of which he suffered at intervals from melancholia and 'paralysis of energy.' About eight months after the partial subsidence of these symptoms, he fell down in the street, unconscious. This was probably of epileptic character, for well-marked epilepsy supervened in a short time, followed by maniacal excitement. An immediate consequence of the first fit was amnesic and heterophasic aphasia. Progressive muscular atrophy, *confined to the right side of the body*, accompanied by considerable loss of power, came on at an uncertain period afterwards. Common sensibility seemed unaffected. Hearing upon right side defective. Articulation also interfered with. Both pupils were persistently contracted. He had epileptic fits at long and irregular intervals, in which the convulsions were limited to the right side of the body. Memory on all points was confused, and talking soon excited him. He died of apoplexy in February, 1874. The post-mortem examination revealed the following appearances. Dura mater adherent. Arachnoid slightly opalescent; small local atrophies in the neighborhood of the intra-parietal fissure. Apoplectic clot in the centre of the right occipital lobe, the size of a walnut; below its level, and on a level with the right corpus striatum, another clot was found, measuring five or six inches in length and about one and a quarter inches in width, extending about an inch from the tip of the frontal lobe to about the same distance from that of the occipital lobe, bounded upon the left by the motor tract, but not implicating it. In the left hemisphere a yellow softening, about the size of a large walnut, was found impinging upon the corpus striatum, involving the extra-ventricular nucleus and claustrum, and extending to within a few lines of the gray matter of the convolutions. The external arteries were much thickened. On the basilar artery large deposits of a yellowish color existed. The middle cerebral arteries in the fissure of Sylvius, on both sides, were seen to be nodulated and rendered moniliform by this deposit." Microscopic examination revealed the fact that the muscular and fibrous coats of the arteries were thickened throughout the brain; surrounding them, in some places, "concentric rings of a material holding corpora amylacea were found. In some

instances empty spaces existed between this material and the brain-substance; in others, this interspace was filled with a colloid-looking substance." "In the pons varolii the vascular canals were in a state of extreme dilatation." "*In both cord and medulla the lesions were symmetrical.*" Many cells had undergone fuscous degeneration; others were bloated and swollen, had lost their angles, and their nuclei and nucleoli were indistinct.

This case is particularly remarkable for the fact that progressive muscular atrophy was developed *unilaterally*: it is the only case of the sort that has been reported, so far as my knowledge extends. Hammond simply alludes to the fact that it may occur; but such cases must be extremely rare. This atrophy, as well as the epileptic attacks, was dependent upon the point of softening in the neighborhood of the corpus striatum; for, you observe, the other cerebral and spinal lesions were *symmetrical*: the unilateral symptoms must have been caused by the unilateral lesion.

As regards the prognosis, I will state that a considerable proportion of cases are amenable to treatment. So long as nerve-tissue is merely irritated, or has its function suppressed by the influence of pressure, its constituent elements remaining intact, you may hope to effect a cure. When ganglionic cells are destroyed, of course you cannot hope that they can be regenerated; yet proper treatment may arrest further destructive processes: although the ship is crippled, she may survive the storms of many seasons and be able to perform much effective work. Hence the prognosis is better than it would be if the morbid nervous phenomena depended upon tubercle or carcinoma for their causation. In a given case, you should reserve your prognosis until you have been able to test by the appropriate remedial measures the amount of damage to nervous structures. If the symptoms are evidently those of irritation, the prospect of cure is much better than when they are those of depression.

This brings us to a consideration of the treatment of syphilitic nervous affections. Those of the earlier stage, which resemble the ordinary neuroses, are evidently due in a great measure to the concomitant anæmia. You must remember that this paucity of red blood-globules does not give way before the administration of ferruginous tonics, differing widely in this respect from the anæmia following hemorrhage, etc. These blood-tonics are required,

certainly, but you must also employ specific medication adapted to the requirements of the case. The protiodide or the corrosive chloride of mercury, given cautiously and for a long period, is generally successfully employed during the primary and secondary stages. Mercurial fumigations, with calomel, have given excellent results in the hands of some. The hypodermic administration of mercury has been essayed; but the local irritation arising from the injections has caused it to be abandoned by most practitioners. If some non-irritating solution of a salt of mercury could be obtained, this method of treatment would offer many advantages over the therapeutic methods now in use.

When nervous affections are developed during the tertiary stage (the sequelæ of syphilis, as we prefer to consider them, agreeing in this with Mr. Jonathan Hutchinson), such as epilepsy, paralysis, and mental impairment, the iodide of potassium in large, even enormous, doses offers the only rational hope of cure. Large doses are borne remarkably well. Dr. Buzzard says it seems to act like a *food* in many cases. Like quinine, the iodide is given with too much caution by many practitioners: they have an unfounded prejudice against giving it in really effective doses. Give twenty-, thirty-, forty-, or even sixty-grain doses every four or six hours. Begin with a moderate amount, say a scruple three times a day; if you see no marked improvement in a week, give more; do not wait until your patient's stomach has become irritable from the smaller amounts, but give it freely, fearlessly, so as to obtain the benefit of the drug at once. Sometimes you may advantageously alternate the iodide with mercury, or may give them in combination. When convulsive seizures of an epileptiform character are frequent, you may often derive excellent results from a combination of the bromide with the iodide of potassium. Combat the ever-present anæmia with chalybeate tonics; add quinine in malarial districts.

In mental disease of specific origin, when insomnia is a prominent symptom, give chloral at night. If there is much agitation and restlessness, conium will probably be of service; but always give the iodide, whatever may be the phase of the affection: it is the remedy *par excellence*.

Last summer, as some of you will recollect, I treated with very gratifying success a case of syphilitic paraplegia, which rapidly

yielded to one hundred and ten grains of the iodide of potassium, daily, given in conjunction with compound tincture of cinchona. The primary galvanic current was applied every second day to the spinal cord, whilst to the paralyzed muscles the induced current was directed. The patient had, prior to our seeing him, almost abandoned all hope of cure. He is now restored, and following his avocation of steamboat clerk.

For a further development of this subject of syphilitic diseases of the nervous system, especially as regards the pathology, diagnosis, and treatment, I refer you to the writings of Lancereau and Moxon, and especially to the recent work of Dr. Buzzard; to an admirable article in the April number, 1874, of the "American Journal of Insanity," embracing in a condensed form the "Lettsomian Lectures," by W. H. Broadbent, M.D., T.R.C.P., and embodying the latest researches upon the subject; also to an excellent article in the "American Journal of Insanity" for January, 1874, entitled "Clinical Observations on the Dementia and Hemiplegia of Syphilis," by M. H. Henry, M.D., Surgeon N. Y. Dispensary.

LECTURE XXXIX.

NEURALGIA.

Definition of Neuralgia.—General Characteristics.—Varieties.—Pain.—Brown-Séquard's Theory.—Woir Mitchell's Theory.—Painful Points of Valleix.—Nerve-Tension.—Intermittence of Pain.—Migraine.—Causes.—Symptoms.—Pathology.—Diagnosis.—Prognosis.—Treatment.—Neuralgia of the Trigemini.—Nerves Affected.—Neuralgic Points.—Causes.—Symptoms.—“Painful Anæsthesia.”—Epileptiform Neuralgia.—Tic Douloureux.—Complications.—Morbid Anatomy and Pathology.—Diagnosis.—Prognosis.—Occipito-Cervical Neuralgia.—Cervico-Brachial Neuralgia.—Intercostal Neuralgia.—Symptoms.—Causes.—Diagnosis.—Painful Points in Intercostal Neuralgia.—Prognosis.—Mastodynia.—Diagnosis.—Zona, or Herpes Zoster.—Lumbo-Abdominal Neuralgia.—Crural Neuralgia.—Sciatica.—Neuralgia of the Sacral Plexus.—Neuralgic Points.—Symptoms.—Causes.—Prognosis.—Treatment of Neuralgia.

GENTLEMEN,—This evening I shall bring to your attention and delineate, so far as the limited time at my command will permit, that most important series of morbid phenomena which, taken together, has received the name of neuralgia. It is very necessary that you should understand the subject, for the affection is among the most common of those “which flesh is heir to,” one which causes the most intense suffering, and which demands immediate relief at the hands of the physician. It is a disease which, if treated energetically and with a proper knowledge of remedial measures, you will often be able to relieve as if by magic, and thus add largely to your professional reputation; but, unfortunately, in too many cases it will prove obstinate and incorrigible, and a source of annoyance to the practitioner as well as of torment to the patient. I shall attempt to give you some practical points which will generally enable you to determine in a short time the prospect of permanent relief in a given case, or whether all therapeutic measures will be useless or of value only as palliatives.

Neuralgia may be defined as pain manifested in the course of a nerve-trunk or referred to its points of distribution, paroxysmal in character, either showing itself without any evident exciting

cause, or out of all proportion to the excitant when one is apparent; and it is usually limited to one lateral half of the body.

There are certain peculiarities common to all varieties of the affection. These are: limited territories painful on pressure,—the painful or neuralgic points of Valleix, who first described them: they are not always present, and have not quite the diagnostic value attributed to them by their discoverer,—and dependence upon certain general conditions of the system for causation. Another characteristic, strongly insisted upon by Anstie, is the tendency of neuralgia to manifest itself first at the periods of life marked by the changes of bodily development and decay,—puberty and the climacteric epochs. I have referred so many times to the transmutation of nervous diseases that you already know that neuralgia is among the affections transmitted from generation to generation, not only as neuralgia, but the graver forms of the neuroses, or even structural changes of degeneration and decay, may thus originate. A deficiency of normal nervous equilibrium, a condition of preternatural instability of nerve-element, is often, nay, generally, if not invariably, the pathological soil where the different forms of nervous affection find their proper nidus, whence they spring, full of destructive power, surcharged with dangerous energy, threatening the worst consequences to the individual and to the human race. Hereditary predisposition is considered by Anstie to be the most important factor in the genesis of neuralgia. Without this morbid ground, prepared by the misfortunes, faults, errors, or sins of his ancestors, it may well be doubted whether any person would ever suffer from the neuroses.

The symptoms of neuralgia depend for their manifestations upon the origin, course, and distribution of the sensiferous fibres of the several cranial and spinal nerves: hence we shall have to classify them according to the nerves and plexuses affected. I shall have time to describe only the principal varieties of the affection, those with which you will most often have to deal in practice. These are migraine, trigeminal, cervico-occipital, cervico-brachial, and intercostal neuralgia, mastodynia, lumbo-abdominal, crural, and sacral or sciatic neuralgia. We shall proceed to study each variety separately, its symptomatology, etiology, pathology, diagnosis, and prognosis, with the treatment appropriate to each condition.

Pain being the essential characteristic of all forms of neuralgia, it may be well to have as definite ideas of its nature as it is possible to arrive at in the present stage of knowledge, before discussing the subject in relation to the phenomena presented when different locations in the human body are affected.

What is pain? It is a sensation received in and perceived by the brain, or the proper cerebral tract devoted to the purpose, when a nerve is irritated beyond the degree necessary to give rise to the normal sensation. In the lecture upon locomotor ataxia I gave you Brown-Séguard's explanation of the fact that any one of the four different forms of common sensation may be abolished and the others remain intact. His explanation was, that there are four kinds of fibres in every sensiferous nerve: one, each, for the conduction of the sensation of temperature, of touch, of tickling, and of pain. This is a very plausible hypothesis; but there is another which explains the phenomena alluded to equally well, and, also, some not made clear by that of Brown-Séguard. This is, that there are divers brain-centres for the reception and appreciation of the different forms of sensation,—different sensory tracts,—and that these may become changed in some way so as not to receive and register the different impressions conducted thither; that the same nerve may conduct a nervous impulse which is appreciated as a distinct sensation according to the different form, degree, or location of the irritation applied. Weir Mitchell illustrates this postulate very beautifully. He says, "It is as if through a single tube were spoken various languages which could be only understood when at its farther end they reached the ear of the hearer native to each form of speech." *

It is well known that when an irritant which is excessive in intensity or abnormal in quality is applied to the distribution of a nerve in its normal condition, pain results. Thus, an excessively brilliant light, an extremely loud sound, or a powerful odor is painful, and, if long continued, may cause the most exquisite suffering. Again, an excitant of sensation, normal in degree, and perhaps pleasurable at first, if too long continued becomes painful. Besides these facts of common experience, there is one other to which I shall refer: namely, if an irritant of any kind or of any

* *Injuries of Nerves, and their Consequences*, p. 41.

appreciable degree of intensity is applied to a nerve-trunk, to any part of a nerve other than at its peripheral distribution, it causes a sensation of pain, and this does not differ in character in any way corresponding with the nature of the irritant. Whenever a nerve-trunk is irritated, the sensation perceived is referred to its distribution.

The painful points of Valleix offer an apparent contradiction to the latter proposition. These points are found where a nerve-trunk changes its level or relations, by emerging from a bony foramen or by piercing a layer of fascia. They are sometimes found at a point where many branches are given off together. Now, pressure over these points produces pain, not in the distribution of the nerve, but at the point where the pressure is applied. This apparent paradox has received several explanations, the most plausible of which is that the nerves distributed to the nerve-sheath (*nervi nervorum*) are in the same hyperæsthetic condition as the other branches, and, being confined and pressed upon by the unyielding margins, are even more sensitive than other filaments of the affected nerve.

To explain the occurrence of pain from an irritant which is normal in intensity and quality may seem a difficult matter; but a simile may serve to make it plainer. You know that the tone produced by the vibration of a string of a musical instrument depends for its pitch not only upon its size and length, but also upon the degree of tension to which it is subjected. Now, you may consider a nerve in the normal degree of tension as comparable to a musical string giving the tone of *C natural*; when the tension is increased, so that the same excitant produces the sensation of pain, the tension of the string will be such as to produce *C* of the octave above. When this condition is carried to the degree that the sensation becomes intolerable, agonizing, it will correspond to the highest note produced when the string is stretched to the utmost extent; further increase in the tensile force, and the nerve is paralyzed,—the wire breaks. Again, the different forms of physical force, heat, light, electricity, etc., have an influence upon the tone produced by a musical string, by raising or lowering its tension. The transmission of nervous influence may be compared to that of a vibration or undulation through a wire.

That the manifestations of pain are intermittent, while the causative lesions are constant, is a fact worthy of some attention. The explanation is to be found in the proposition that there is a certain intermittence, a rhythm, in all organic processes; and this is especially evident in the phenomena presented by the nervous system. The clonic convulsion which marks the sudden discharge of nerve-force is the motor counterpart of the intermittent pain of neuralgia, which denotes the transmission of an irritative impression along a nerve to its centre. In the one case, the nerve-force continues to be discharged until all that is available is exhausted, and there must be a period of rest, of recuperation, until more can be generated; in the other, the nervous impulse is transmitted along its conductor until it is unfitted for this function, and can no longer conduct; then there must be a period of quiescence before the molecules can be reinstated in their normal condition and the nerve resume its function.

The first variety of hyperæsthesia which claims our attention is that known to every one by the term sick-headache; in medical literature it is called *migraine*, or hemicrania. Some authors deny that it is a variety of neuralgia, because many characteristics of the latter-named affection are absent. Many are, however, present: so, for convenience of classification, as well as from its causation and clinical history, we shall consider it in this connection.

Constitutional predisposition is the underlying fact in probably all cases of migraine. Age is another important factor in the initiation of the affection. As I told you when speaking of tubercular meningitis, all forms of neuralgia are extremely rare in children: therefore you need never look for this affection before the completion of the second dentition. This form generally makes its first appearance during the developmental processes, which are completed before the twentieth year. This is especially true of females, in whom there is such an immense tax levied upon all the energies of the system at the period of puberty. If, while the girl is thus subjected to unusual strain, the intellectual faculties are unduly stimulated,—if proper allowance is not made for the periodical demands upon her nervous system,—if, as is unhappily too often the case, she is allowed to participate in the excitements of the ball, the theatre, and society, in addition

to competition in her studies with her fellows, as well as with members of the opposite sex,—then may we expect not only neuralgias, hysteria, and chorea to be developed, but also insanity of the most hopeless kind, should she be subjected to any great moral or physical shock. Another kind of social condition is equally dangerous to the child approaching the great change of puberty. Wherever this demand upon the system is accompanied by physical over-work and insufficient or improper nutrition, migraine is very apt to be developed. Many women suffer from sick-headache just before or during the time of every menstrual flow. In some cases it may be traced to uterine displacements or inflammations. In malarial districts this is a common form of the manifestation of a masked ague. The exciting causes are mental excitement or over-work, loss of sleep, prolonged use of the eyes, exposure to the direct rays of the sun, etc.

Migraine generally commences in this way. A person rises in the morning with an undefinable feeling of discomfort, of lassitude, want of appetite, and with a slimy, disagreeable taste in the mouth. He has slight chilly sensations and yawning. The first vague feeling of discomfort rapidly passes into one of decided pain. Its unilateral character may not be perceived during the first attacks; but when he has become accustomed to observe himself closely, this fact of the limitation of the pain to one side of the head is generally apparent; occasionally, however, both sides are affected alike. When it is unilateral, the left is commonly the side involved. The suffering soon becomes almost unbearable; the patient flies from all disturbing influences; the bodily strength seems almost abolished, and he seeks his couch, not for repose, for it comes not, but because of the failure of his muscular power. The optic and aural nerves are involved in the hyperæsthesia; light and sound are alike intolerable. There is great tenderness over the scalp, and even touching the hair is painful. Sometimes the superficial tenderness is clearly limited; or the pain may be confined to circumscribed points,—to the parietal, a little above the eminence of that name, or to the supra-orbital, over that notch or foramen. This localization of the headache gives the idea of a nail being driven into the skull: hence the name *clavus*. The adjective generally affixed, *hystericus*, forms an unfortunate conjunction of words; for these limited painful points are found in

women who have no uterine disease, and also in patients of the opposite sex.

The lachrymal, pituitary, and salivary secretions are often augmented, showing a paralytic condition of the vaso-motor, or, according to the recent researches of Vulpian, an increased excitation of the secretory nerves of the glands and membranes affected. Nausea and vomiting generally supervene after the headache has continued several hours. Large quantities of acrid, greenish-yellow or brownish materials are ejected, with much retching. If food has been taken just before the attack, it remains undigested, and is ejected along with the gastric secretions and regurgitated bile. There is evidently a hypersecretion of the latter fluid, corresponding with that of the others mentioned. It should be observed that indigestion has nothing to do with the inception of the attack.

The duration of one access of hemicrania varies from twelve to twenty-four hours, rarely passing this limit. The vomiting is generally the signal for the headache to cease. Sometimes patients attempt to hasten this event by irritating the fauces; but emesis excited in this way has seldom any beneficial effect. After the headache passes away, the patient usually sleeps, and the next day has regained his ordinary health, with the exception of some weakness owing to want of food and rest.

I should have observed that in the early part of the attack the surface is pale and cool, and the pulse increased in frequency. The temporal artery will be found small and hard under the finger. Later, these conditions are reversed, the conjunctivæ are reddened, the face is flushed, and sometimes there is unilateral sweating of the face.

Morbid anatomy teaches us nothing regarding migraine: the morbid process leaves no trace behind. There is undoubtedly a functional change in the branches of the fifth pair of cranial nerves distributed to the meninges and to the structures of the side of the head, as well as in the fibres of the sympathetic going to the cranial arteries and arterioles.

The use of the thermometer will enable you to exclude all suspicion of meningitis in first attacks, the temperature being normal in all forms of neuralgia, and as uniformly elevated in inflammations. The main point in diagnosis is to determine the cause.

Hence you will look for menstrual derangements, for uterine deviations and inflammations, for exhausting discharges, for rheumatism, gout, syphilis, and for evidences of malarial poisoning.

Cases clearly traceable to malaria are the most hopeful. If syphilis or anæmia is ascertained to be the cause, you can generally promise relief. If the uterus is at fault, the climacteric period may end your patient's suffering from hemicrania. Hence the prognosis depends upon the cause, and upon the facility of its removal. In a large proportion of cases you will be limited in your prognosis to the opinion that if the general health is kept up to the highest standard the attacks will be reduced to the minimum of frequency,—*not* entirely prevented.

You will address your therapeutic measures to the removal of the cause, whenever this can be ascertained. Neuralgic patients are often the victims of chronic constipation. *Nux vomica* and its alkaloids are very useful in combating this condition. General conditions, such as the different diatheses and malarial poisoning, are to be treated by appropriate remedies, of which I shall speak at length when we consider the treatment of neuralgia in general.

During the attack the patient must be kept perfectly quiet in a darkened room. No food is to be administered: let him drink iced Seltzer or Vichy water, or lemonade. A large dose of bromide of potassium, especially if it is combined with chloral hydrate or croton chloral, may abort the paroxysm if taken in the initiatory stage. Citrate of caffein in doses of gr. i or gr. ij, repeated every hour, may shorten the attack if given early. *Paulinia sorbilis* (guarana) may be given in doses of gr. vi–x of the extract. It may prove of some service; but I have been disappointed in its effects. But the best and most certain of the measures adapted to remove the disease when it is fully developed is galvanization of the cervical sympathetic. Firm pressure with the fingers upon the carotids will generally give temporary relief, but it must not be too long continued; and you must be extremely careful about employing it if your patient is past the meridian of life, for rupture of a cerebral artery might result. Rubbing the eyelids with extract of belladonna has given partial relief in some cases. The hypodermic injection of a solution of morphia or of atropia may be urgently demanded; relief must be had at any price, in some aggravated cases, and, if you hesitate, some more

complaisant practitioner will relieve you of your case. The sixth of a grain of morphine, or the one-hundred-and-twentieth or one-hundredth of a grain of atropine, will be sufficient in most cases. I earnestly warn you against giving a larger dose until you have ascertained how these drugs are borne. The administration of morphia often increases the nausea and nervousness after the headache has passed away, and in some cases atropine causes such distressing head-troubles that you are unable to give an effective dose. In most cases the patient suffers least if he goes to bed and eschews all medication during the attack, directing his attention to the improvement of his general health and the avoidance of exciting causes in the intervals between the attacks.

I told you that in hemicrania the nerves in a hyperæsthetic condition are some of the branches of the fifth pair. We shall now begin the consideration of a most distressing affection,—neuralgia limited to the sensory branches of the trigeminus. In migraine there is some possibility of doubt, in many cases, as regards the nerve-fibres affected; but in face-ache (of Fothergill) the correspondence of the location of the pain with the known anatomical distribution of the branches of the trigeminus forbids any discussion as to its seat. Next to sciatica, this is the most common variety of neuralgia to be met with in practice; one which will most often prove rebellious to treatment; one in which your therapeutic measures must too often be limited to simply palliating the evil which you are powerless to relieve effectively and permanently.

The second, or superior maxillary division, is most often the seat of the hyperæsthesia. The pain is then referred to the alæ of the nose, to the cheek, upper lip, and teeth, and to the depths of the palatine and nasal fossæ. The ophthalmic division is affected next in order of frequency: in malarial localities the supra-orbital branch is the seat of pain more often than any other part of the trigeminus; this is what is termed “brow-ague” by the common people. The pain may be referred to the forehead, eyebrow, inner angle of the eye, caruncula lachrymalis, upper eyelid, or the deeper parts of the orbit. The inferior maxillary is the division least often affected. The pain is then felt in the lower lip and teeth and along the border of the lower jaw.

In well-developed cases you will find many points upon the

surface painful on pressure, but the principal of these are to be found nearly upon a vertical line, and correspond to the points of emergence of the three divisions of the trifacial nerve from the bony canals. These are over the supra-orbital, the infra-orbital, and the mental foramina.

Neuralgia of the fifth pair of cranial nerves is a type of the disease wherever found: hence what I shall say of its causation, diagnosis, and pathology applies in the main to all varieties of the affection. Of course there are some special causes of the painful affection peculiar to certain regions; these it will be my duty to refer to in connection with the disease appearing in certain locations.

The causes of trifacial neuralgia, and of all other varieties, may be divided into constitutional and local.

In a very large proportion of cases there is a neurotic temperament or predisposition. This tendency to nervous affections cannot be foretold in a given case, except by closely examining the individual and ascertaining his complete family history. You will find the latter procedure impossible in a large number of cases which you will meet in practice. In some patients the general appearance, countenance, and manner will set you upon the right track; in other neurasthenic persons you will observe only the appearance of general good health, and the tendency to the neuroses will be made apparent only by their development under the stress of adverse circumstances. As you cannot tell from its appearance whether an apple is sour or sweet, or whether a new chemical compound is an acid or an alkali, so you cannot single out your future neuralgic patient in a crowd of healthy people. Exposure to depressing causes, and the changes of development or of decay in the bodily organism, these are the tests of his liability to this, as well as to many other nervous affections to which I have called your attention. Among the constitutional causes are to be reckoned the syphilitic, rheumatic, and gouty diatheses, chloro-anæmia, and malarial, mercurial, saturnine, and cuprous poisoning; the suppression of habitual discharges,—*e.g.*, menstrual or hemorrhoidal; the persistence of exhausting discharges,—*e.g.*, menorrhagia, leucorrhœa, etc.

The local causes are exposure to cold, particularly to draughts of air; pressure upon a nerve-trunk by tumors, exostoses, or foreign

bodies; carious teeth; cicatricial contractions following wounds or burns; and disease of the antrum of Highmore. Caries of the petrous portion of the temporal bone and pressure upon the nerve between its point of origin and entrance into its bony canal by intra-cranial new formations, such as the products of meningitis, deposits of tubercle, cancerous or syphilitic masses,—each of these may occasion neuralgia of the trigeminus.

Before the outbreak of an attack of trifacial neuralgia, there are sometimes certain prodromata which indicate the coming on of the storm. There may be only a soreness upon pressure, a tenderness over the painful points of Valleix; or there may be shooting pains, slight in character, but which are only too well understood by the habitual sufferer from neuralgia. After the attack is fully developed, pressure over these points excites a paroxysm of pain throughout the affected district, and is very painful at the point pressed upon. In some cases, if the pressure is firmly applied and long continued, it has the effect of giving temporary relief; in others, cutaneous anæsthesia may be induced by the pressure interfering with the transmission of external impressions, yet the pain will remain as great as before, and still be referred to the surface. This is the “painful anæsthesia” of some authors. The attacks of neuralgia are made up of paroxysms of greater or less intensity. The paroxysms may be very near together, a few minutes, or a few seconds, intervening; the pain is usually slight at first, but rapidly increases in intensity until its height is reached, when it declines in the same way. The attack usually consists of a number of these paroxysms, which gradually decrease in intensity and frequency until they cease entirely. The attacks are apt to be intermittent or periodical. This is markedly observable when they owe their origin to malaria.

There is a special form of neuralgia, described by the illustrious Trousseau under the name of *tic épileptiforme*, so called from some resemblance which it bears to the epileptic aura. This variety generally is limited to the trifacial nerve; if any other is involved it is usually the great occipital. The paroxysm comes on without warning, is of *frightful severity* from the first, lasts but a few seconds or minutes, and as instantly ceases. It thus bears some resemblance to the electric pains of locomotor ataxia. The paroxysm is lighted up by the slightest excitant: a breath of air, the contact

of a feather, the act of masticating the food, etc. Jaccoud relates a case where the first word spoken in the morning, or the contact of a drinking-vessel with the lips, was sufficient to initiate a day of suffering; the patient's nights were passed very comfortably.

During the attack the features are convulsed; the motor nerves supplying the muscles of expression, the *portio dura* of the seventh pair, receive a reflex irritation, which thus manifests itself. Sometimes other motor centres may become involved in this reflex irritation: thus the muscles of the opposite side of the face involuntarily contract; and, what is very uncommon, those of the trunk and extremities may participate in the convulsive action. This tonic spasm of the muscles of expression has given rise to a common name applied to the disease as a whole,—another instance of naming a disease from one symptom, which I have had occasion to deplore in relation to so many of the affections which I have brought to your attention. *Tic douloureux* is as familiar in popular phraseology as is apoplexy or paralysis. The term *tic* refers to this involuntary contraction of the facial muscles, resembling a trick or habit of expression peculiar to certain persons. This form of neuralgia is not amenable to remedial measures: it may be palliated, but it is practically incurable. It appears during middle life or old age, and is often the precursor of epilepsy or the grave forms of nervous degeneration. During the paroxysm the patient presses his hand against his cheek, often rubs the skin until the beard is removed, moans and groans with the suppressed agony, paces up and down the room like a caged wild beast, or throws himself upon his bed or the ground and cries out with the extremity of the torture he endures. He ransacks his memory and tasks his imagination to find words sufficiently expressive to convey to others an idea of the torment he endures.

At first, whatever may be the cause of the neuralgia, he has intervals of complete rest, of respite from his sufferings: but, after the disease has continued for weeks, it may be for months, a persistent soreness, or a dull, heavy pain, remains during the intervals: the neuralgia has become continuous, so far as it is possible for a paroxysmal affection to be such.

Trifacial neuralgia is often accompanied by very important complications. Changes of a trophic character occur, which are a source not only of annoyance to the poor sufferer, but sometimes

also of real danger. Such are the changes which may take place in the eye,—iritis, conjunctivitis, and keratitis: they are markedly characteristic, and any one of them may result in loss of sight. Anstie considers all cases of so-called rheumatic iritis to be really of neuralgic origin. Inflammation, apparently of an erysipelatous character, may attack the integuments covering the affected side of the face. Herpetic eruptions appear about the lips and cheek of the same side. The hair of the scalp, eyebrows, and beard may fall, or the roots may become hypertrophied. In other cases grayness may result; and, what is rather remarkable, it may be very transitory, disappearing in a few days after the attack has passed away, *without any of the hair falling*, as Anstie relates in his own case,—he having been a life-long sufferer. In certain rare cases, the skin over the part to which the affected nerve is distributed becomes thinned in texture and glistening in appearance; in other cases, still more exceptional, the subcutaneous tissue becomes thickened, and the result is permanent deformity.

The vaso-motor disturbance is shown by increased secretion of saliva, tears, and nasal mucus. The conjunctiva is reddened; the temporal artery feels large under the finger; the temperature locally is heightened; and the sudoriparous glands pour out an increased secretion, resulting in some cases in unilateral sweating.

When a neuralgic patient dies, it is generally of some inter-current disease; the deaths from neuralgia are very rare, and to be referred to the causative affection rather than to neuralgia *per se*.

Some opportunities have thus been afforded of observing the morbid anatomy. In some cases the neurilemma has been found thickened or distended with the products of ordinary inflammation, or, in syphilitic cases, with the gummy material of which I spoke in my last lecture. The nerve-trunk has been found stretched or flattened by the pressure of tumors, exostoses, or foreign bodies. The nerve-cells about the nucleus, or point of deep origin, have been observed atrophied, or in a state of fatty or pigmentary degeneration. We may be allowed to conjecture, where no post-mortem lesions can be detected, that the morbid condition was that of a hyperæmia of the nerve-sheath, which, like the other forms of congestion that I have described to you, disappears soon after the cessation of cardiac action. However,

no one distinctive lesion has been found which clearly indicates to the eye of the practiced microscopist the essential change to which might be referred the distinctive symptom of *pain* in the course or distribution of a nerve.

That changes do occur we cannot doubt. That there is a condition of hyperæmia of the centre for the nerve affected may well be received with much reserve; although this hypothesis has had many supporters. That there is a condition of irritation is plain to the casual observer. That this condition coincides with anæmia of the central nuclei is held by Radcliffe, Anstie, and most of the latest and best authorities upon the subject, and derives much support from the modes of causation, when known, as well as from the results of treatment in the vast majority of cases. That neuralgia is closely connected with the graver forms of nervous disease—graver in that they are more immediately dangerous to life or reason—is proven by the fact that epilepsy or insanity often appears in the later stages, especially in that form which first appears late in life, the neuralgia being really merely a prodromic symptom of the later affection. Now, it is pretty well proven, as I stated in a former lecture, that insanity has for its basis an irritated condition of the cortical cells, which have for their function the manifestation of mental phenomena; this irritation goes on to produce degenerative changes easily verified by microscopic examination. Certain psychical symptoms, usually of a depressive, melancholic character, which may eventuate in suicide, appear in quite a large proportion of sufferers from the graver forms of trifacial neuralgia. Hence these mental phenomena possess a certain degree of pathological significance.

Trifacial neuralgia—sometimes called prosopalgia—is to be discriminated from odontalgia (toothache) by its strictly paroxysmal character and by the presence of painful points in the regions before indicated. This diagnosis it is very important to make, for mistakes of this character are common, and you will find many of these patients who have parted with tooth after tooth until all have been removed without improving their condition in the least. To ascertain the cause is the important point in order to give a diagnosis of any practical value. In relation to neuralgia this is of paramount importance, for here we have to deal with many possible factors, and the prognosis as well as the treatment is to

be based upon the etiology. The diagnosis which is confined simply and solely to the detection of the pathological changes which present themselves on examination is practically useless as regards the therapeutics of any disease. This kind of diagnosis is good enough for the study of the medical philosopher ; it will enable him to construct nosological tables, compute statistics, and show to the world the extent and profundity of his learning ; but, as practical physicians, you will require something more than this to enable you to do battle successfully with disease and to be of benefit to the human race. You must know the cause of a given morbid condition, in order that you may be able to adopt prophylactic measures,—often far more valuable than those addressed to the cure of the affection,—and also that you may be able to go to the root of the matter and lend a hand in its extirpation ; in other words, to treat it not only palliatively, but also with a view to its permanent relief.

You will gather as complete a history of the case as you possibly can ; you will consider the fact whether your patient is at present, or has been recently, a resident of a malarious district or not ; you will ask if there is any periodicity of the attacks, if he has ever had ague, or if he has been subject to any exhausting discharge. Examine the heart and cervical blood-vessels for the hæmic murmurs present in anæmia ; look at the tongue for marks made by the teeth upon this member in such a condition of the system ; also at the lips for pallor. Look for evidences of syphilis upon the skin, in the throat and mouth, upon the superficial bones, and in the glands of the groins and the post-cervical region. Ascertain if his family history shows the presence of a gouty or rheumatic diathesis, and if he has ever suffered from any articular disease ; exposure to humidity or to draughts of air will naturally occur to your mind in this relation.

Look for dental caries, for inflammation in the antrum, for cicatrices, and for otorrhœa. Any tumor or swelling in the parts traversed by the nervous branches will immediately attract your attention. Remember the fact that neuralgia of the trigeminus, or of any other nerve, may be caused by a nervous injury in any part of the body, however far removed : that is to say, it may be of reflex origin.

I have been thus particular in considering the diagnosis of tri-

facial neuralgia in relation to its causation, for the reason that the same or similar procedures are necessary in the clinical study of each of the varieties we have yet to examine.

The causation determines to a great extent the prognosis. If it be of malarial origin, you may promise relief in almost every case. If it be due to anæmia, the same may be said. If syphilis, rheumatism, or gout be at the basis, it is possible in about half the cases to effect relief for some time; but you must expect oft-repeated relapses. If decayed teeth, foreign bodies impacted, or tumors which can be removed be present, there is a strong probability that removal of the cause will abate the disease; but remember that if it have continued long a morbid *habit* may have become established, which often causes the hyperæsthetic condition to remain after the removal of the irritating body. Thus you see that the prognosis is uncertain in nearly every case. This will be more evident when you consider that in almost every instance there is a constitutional predisposition present which cannot be removed by remedies. In those cases in which you find the pain persisting between the attacks, your prognosis must be very guarded: there is generally a permanent source of irritation, such as a tumor or an exostosis, pressing upon the nerve-trunk, and deeply seated. Where you find the condition which I described to you as "painful anæsthesia," you may be certain that there is some irritant applied to the nerve-trunk, either within the cranium or while it is still within its bony canal, and cannot, therefore, give your patient much encouragement.

Niemeyer points out the fact that a case is grave in proportion to the number of branches involved: thus, if the pain is confined to one small branch, it shows that the point of irritation is comparatively superficial, while if all the branches of one division are affected, and more especially if all three divisions are alike involved, the excitant of pain must be profoundly situated, and hence there is less probability of reaching it effectively. The epileptiform variety is absolutely hopeless as regards permanent relief: you may palliate, but that is all.

Neuralgia appearing at or about the time of puberty you have a good prospect of relieving, but you will be morally certain that any depressing circumstance in after-life will reproduce it in all its former intensity.

The treatment appropriate to each variety of this affection we shall consider at the end of the lecture.

In the next variety which we shall consider,—the cervico-occipital,—the sensory branches of the first four cervical nerves are affected. It is quite uncommon, and exposure to cold and moisture is usually the cause. The symptoms are pain in the neck and side of the head, often shooting into the face; the inferior maxillary branch of the fifth cranial often participates in the hyperæsthesia. The pain is intermittent, but in the intervals there is felt a deep-seated pain in the back of the neck. The attacks are often very severe, and are excited by the slightest cause: a breath of cold air, contact with clothing, or even with a lock of hair, will often induce them.

In cervico-brachial neuralgia the nerves affected are the sensory fibres of the four lower cervical and the first dorsal. The pain is felt in the shoulder, arm, axilla, fore-arm, and fingers. It comes on in paroxysms of acute, lancinating, or burning pain, following the course of the principal nerves. During the intervals a dull pain, or a burning, tingling, or numb sensation, is felt throughout the member.

The principal causes are over-exertion of the arm, rheumatism (which does not brighten the prognosis), punctured wounds of nervous twigs in venesection, impaction of foreign bodies (*e.g.*, projectiles) near the nerve-trunks, and disease of the vertebræ. Aneurisms in the axilla, and cancerous growths, have given rise to some extremely obstinate cases in this region. Like the last described, it is rare, and the cause can seldom be traced to malaria.

The next variety to engage our attention is, practically, of considerable importance, because of the liability to make mistakes in diagnosis. I allude to intercostal neuralgia. The intercostal nerves are the ones affected.

The paroxysmal character of the pain is not so well marked as in the varieties which we have been studying. Darting, lancinating pains, excited by movement or pressure, are what cause the patient to apply to you for relief. Respiration excites these pains, so that they appear to be almost continuous. The clothing has to be worn very loose, in order to avert pressure from the painful points. A feeling of constriction, or, usually, of semi-constriction, accompanies the pains. The patient limits the action of all the

respiratory muscles, except the diaphragm, as much as possible. He dare not cough or sneeze,—acting, for all the world, like a patient suffering from acute pleuritis. This he—or she, for three-fourths of the cases occur in females—is believed to have, both by himself and by his friends; and thoughtless and careless physicians have often made the same error in diagnosis, excusable in the laity, but unpardonable in a practitioner of our noble art.

Women are especially liable to this form of neuralgia. It is commonly developed with the anæmia which accompanies disordered menstruation, leucorrhœa, uterine catarrh, or malarial poisoning. Hemorrhage from any source, exhausting discharges of all kinds, the callus formed about the ends of a broken rib, disease of the vertebræ, and the pressure exerted by carcinomatous and aneurismal tumors, may be, any one of them, a cause of this form of neuralgia.

You will often meet with patients suffering from this disease who are firmly convinced that they have pleuritis, phthisis, or hepatic disease (“liver-complaint”). Auscultation and percussion will enable you to exclude these affections, and the finding of the painful points peculiar to this form of neuralgia will make your diagnosis complete. These points are invariably present, and are very easily detected. You are to look for them in three different situations, corresponding to the points of emergence of the nerve from the inter-vertebral foramen, of the middle perforating branch, and of its anterior perforating branch or terminal distribution. These are at the angle of the rib and over the spinous processes of the dorsal vertebra,—always present; on a line let fall from the axilla,—often wanting; and at the outer border of the sternum in the intercostal space,—always present. The slightest pressure over these points, especially the last named, causes the patient to wince or cry out. Of course there may be phthisis present as well as intercostal neuralgia. Happily, you will very often be able to relieve the latter affection by the tonic and reconstructive measures adopted for the former, and thus eliminate one source of suffering.

Your prognosis is to be based upon the causation. If the cause can be removed (and this is usually the case), you can promise relief. Those caused by incurable lesions are necessarily hopeless: you can palliate, but will not be able to cure.

In women, in those suffering from leucorrhœa, there is often complaint of a painful point just below the nipple of the left mamma. Why it should be almost entirely limited to the left side is without rational explanation. A painful knot or nodule is generally detected on careful examination. This is not a neuroma, but is composed of condensed cellular tissue. There may be great fear, amounting almost to hypochondriasis, of heart-disease, or of the formation of mammary cancer, on the part of your patient. The existence of the usual painful points of intercostal neuralgia, the presence of leucorrhœa and of tenderness over the dorsal vertebræ, the fact that the nodule is movable and does not involve the glandular structure, and the absence of valvular murmurs other than those dependent upon anæmia, will enable you to make the proper diagnosis and to give a cheerful prognosis.

In connection with neuralgia of the intercostal nerves, there sometimes occurs an eruption of herpetic vesicles located as exactly over the course of a nerve-trunk as if a practiced anatomist had mapped it out. This eruption is unilateral, runs a certain course uninfluenced by remedies, and seldom returns. The neuralgic pain precedes, accompanies, and follows the eruption, which is of two or three weeks' duration. It is caused by exposure to cold, or by suppression of the menses, or it may appear first at the menopause; it is sometimes followed by obstinate ulcers, which are exquisitely painful to the touch. Zoster is a true neurosis of the skin, and its connection with neuralgia is apparent to an observer who has some knowledge of anatomy. The disease being self-limited, treatment is to be directed to allaying the local irritation. In very rare cases it runs a chronic course; it is then a source of great suffering, and is incurable.

Neuralgia affecting the sensory branches of the lumbar plexus is far from uncommon. The pain is constant, with sharp exacerbations. It is referred to the anterior walls of the abdomen, the external genitals, the lower part of the back, and the buttocks. It has the same causes as intercostal neuralgia, and the remarks concerning the diagnosis and prognosis of that affection are applicable to this variety.

The lumbar plexus gives off sensory fibres which are distributed to the anterior surface of the thigh and leg, and to the dorsum of the foot. These fibres, in rare cases, are the seat of neuralgic

pain. Malaria is probably the most common cause. The symptoms are, pain referred to the points of distribution of these branches, increased by muscular exertion, and often accompanied by cramps (tonic spasms) in those muscles which receive their motor impulse from that plexus. The etiology, prognosis, and treatment are similar to those of the next variety, the last that I shall consider this evening.

The sacral plexus consists of the two last lumbar and two first sacral nerves. Neuralgia affecting any of the sensory branches arising from this plexus is termed sciatica. As Romberg remarks, there is no cutaneous nerve-twigg from the hip to the ends of the toes which may not be affected with sciatic neuralgia,—tradition alone locating it in the sciatic nerve exclusively. Hence a person may have sciatica, and yet the great nerve of that name, with all its branches, remain free from hyperæsthesia.

The painful points of Valleix are very numerous. I shall indicate only a few of the more important. One is situated over the sciatic notch, midway between the great trochanter and the tuberosity of the ischium; it is over the point of emergence of the great sciatic, as well as of the inferior gluteal, nerve; it is the one most easy of detection. Others are located over the superior articulation of the tibia with the fibula, and just back of the external malleolus.

Pain may be felt in any of the sensory branches of this plexus. It is most commonly experienced in the outer part of the thigh, along the back of the leg, heel, and sole of the foot. The pain is continuous, and subject to exacerbations from movements, coughing, sneezing, or standing upon the affected limb. The patient favors the painful member, which, if the disease continue long, notably diminishes in size. The irritation is reflected to the motor centres, and extremely painful cramps (tonic spasms) appear in the muscles of the thigh and calf. The paroxysms often come on after the patient has lain down to rest,—when he is driven from his bed by the severity of his suffering.

This form of neuralgia is, next to migraine, the one most often met with in practice. It is generally extremely obstinate, and its average duration is to be computed by months, if not years.

Males are more subject to it than females: it is an affection of middle life, from twenty to sixty years of age. Rheumatism is

the most common predisposing cause, and exposure to wet and cold is the most ordinary excitant of the attack. Malaria is the etiological factor next in order of frequency. Pressure of the child's head in the latter months of pregnancy, and particularly during a protracted labor, often gives rise to distressing pains, accompanied by cramps. Peri-uterine inflammation, a retroverted womb, or one which is enlarged by fibroid or carcinomatous growths, may produce sciatica of great intensity and long duration. Impacted fæces, or the irritation of the sacral plexus by the passage over it of hardened, desiccated fæcal masses, which occurs in obstinate constipation, is, as Chambers remarks, a common mode of origin. The irritation of tight boots, aneurismal or carcinomatous tumors in any part of the limb, the pressure of a foreign body impacted near a nervous branch, or the pricking of a nerve-twigg in the performance of venesection about the foot or ankle, fractured bones, disease in the hip-, knee-, or ankle-joint, as well as syphilitic nodes, may, any of them, produce sciatic neuralgia.

The diagnosis is limited to a recognition of the cause: on this point I have already dwelt at length. The prognosis is uncertain in every case; but there is a good prospect of relief, which will be lasting in those dependent upon pregnancy or malaria, while relapses are almost certain to occur where the condition originates in rheumatism, or when it first appears late in life.

We have now reached the consideration of the treatment of neuralgia, whatever may be its location.

Remedial measures, in this relation, may be divided into those to be made use of during the attack, which are merely palliative in design; and those to be employed during the intervals,—general or constitutional remedies, which are intended to remove the morbid conditions upon which the pain depends.

As is the case with all obstinate and intractable diseases, a host of remedies have been vaunted as specifics for neuralgia. However, I shall not burden your memory with a catalogue of the different medicines which have been claimed to be efficient in combating the affection and have enjoyed a short-lived reputation only to sink into merited oblivion. The measures that I shall specify have sustained the test of experience, and you can trust to them.

The first indication is to remove the pain. The different preparations of opium are urgently demanded for the fulfillment of this indication. In trifacial neuralgia, the sixth of a grain of morphine administered hypodermically is generally sufficient to give almost instant relief. In the epileptiform variety, Trousseau recommends the bold and free administration of opium, in extract or substance. It is to be given in large, increasing, and oft-repeated doses, until the pain is annulled, or until the physiological effects of the drug are apparent. In most cases large, even enormous, doses are taken without any deleterious consequences, and with the effect of allaying the paroxysms for a time,—weeks or months,—but with no hope of permanent cure. The excision of a portion of the nerve-trunk is generally followed by complete relief for a varying period, but the pain returns in some other branch, sooner or later. Nélaton and Carnochan have reported cases where the relief was of two or more years' duration. In some exceptional cases, opium produces distressing symptoms,—diarrhoea, faintness, nausea, and vomiting. Lotions containing cyanide of potassium, aconite, veratria, or chloroform may be applied with some temporary benefit.

In neuralgic affections of the mammæ and the pelvic organs, belladonna and its alkaloid seem to have some special influence. Plasters and ointments of the extract, or, preferably, hypodermic injections of a solution of atropine, may be essayed, with a fair prospect of giving immediate relief. In the use of the latter, never begin with a dose larger than one-hundred-and-twentieth to one-hundredth of a grain. Many patients are unable to tolerate an effective dose, because of the alarming head-symptoms produced. There is no difference in the effects of medicines administered hypodermically in relation to the point at which they are introduced into the system: the action is upon the economy in general, not in any degree localized. You will, therefore, select the most convenient point for their introduction.

Between the attacks we must endeavor to remove the cause. To combat the neurotic diathesis certain hygienic and dietetic measures are to be adopted. These are: fresh air, out-door exercise, plenty of sunlight, and a diet containing a good proportion of *fat*; the latter, especially, is to be insisted upon. You know that fat is an indispensable constituent of nervous tissue. Starch

and sugar, although hydrocarbonaceous, and capable of undergoing a change into fat in the system, do not furnish the proper pabulum to the nervous structures. Radcliffe and Anstie insist strongly upon this point. Hence you will give cod-liver oil freely. If this is not well borne, let your neuralgic patients have large quantities of fresh cream. Phosphorus may be given, finely divided, in pill form, in doses of one-hundredth to one-sixtieth of a grain, repeated three or four times daily. Several obstinate cases of trifacial and sciatic neuralgia have yielded to phosphorus after various other remedies have been tried in vain.

When the painful affection can be traced to anæmia, ferruginous preparations, with bitter tonics, will often produce brilliant results. In the earlier stages of syphilis, combine mercury with tonics; later, give iodide of potassium, boldly and freely, with or without mercury, paying attention to the stage of the disease. Tonics are always required.

When there is evidence of malarial poisoning, or when this is not suspected, but a marked periodicity is manifested by the symptoms, you will give quinine in large and repeated doses. I will mention one fact which experience has taught me in this relation, which is contrary to the great authority of Anstie. That is, it is necessary to give quinine in much larger doses and much larger quantity to relieve neuralgia than are required to cure ordinary intermittent or remittent fever. Should quinine fail, give arsenic. Fowler's solution may be given, by the mouth, in gradually-increasing doses, or by subcutaneous injection. If you use the latter mode, you may begin with four drops, three times a day, diluted with three times the quantity of distilled water. Of course, great care is to be taken, however used, to avoid its toxic effects. By giving it only after meals, and occasionally suspending treatment for three or four days, you will seldom have any trouble.

Where you find rheumatism or gout present, give the iodide of potassium, in combination with colchicum. These medicines are especially useful in cervico-brachial and sciatic neuralgia. In the intercostal variety, and in mastodynia, give muriate of ammonia, iron, and quinine. Of course, all exhausting discharges and uterine diseases are to receive their appropriate treatment.

In sciatica, in addition to the anti-rheumatic measures proper to the case, acupuncture, with hypodermic injection of a minute

quantity of a very strong solution of morphine, has proved remarkably successful in the skillful hands of Hammond. He uses Lawson's solution of morphine (gr. x. ad aquæ destil., f 5ij. M. Solvet. Two minims—one-sixth of a grain of morphine—are the initial dose; this may be increased to half a grain, if necessary. He prefers to inject this into the nerve-sheath. His manner of procedure is as follows: "Select a point on the posterior aspect of the thigh, about four inches below the trochanter major of the femur, and an inch exterior to the median line; push the point of the syringe perpendicularly, and with no great haste, and at a depth varying from one to two inches you will strike the nerve. You will know this by a slight thrilling sensation passing down the patient's limb. All you have to do now is to inject and withdraw the syringe."*

To avoid pain, he advises local anaesthesia, at the point to be punctured, by the use of ether spray. He says that the procedure is not only palliative, but also curative. A mixture of oil of turpentine (f 5j) with castor oil (f 5ij) has given remarkable relief in some cases. Chambers thinks this is to be explained by its counter-irritant effect upon the sacral plexus at the point where the rectum is in contact with it. Chambers gives much larger doses of the turpentine than that indicated above,—as high as f 3ss every second day: but this is unnecessary.

Croton chloral may be given, more particularly in facial neuralgia: it has more anodyne effect than the chloral hydrate, which is simply a hypnotic. It is a remarkable fact that when given in small doses it produces anaesthesia of the fifth nerve, singling out one nerve, and that one alone, while the sensibility of the body generally, and pulse and respiration, remain unaffected. Opium and its preparations are the most efficient remedies for the paroxysm. Electricity, in its various forms, is the agent *par excellence* to be used both during the paroxysmal recurrences and the periods of intermission of all neuralgic attacks. Faradization has its advocates; but according to my experience, nothing equals the magic effect of the primary galvanic current of *moderate intensity*. The *anode* should be applied over the *seat of pain*, especially over the point of emergence of the affected nerve, and the cathode should

* Clinical Lectures on Diseases of the Nervous System, p. 271.

be placed at some distant point and in such a manner as to favor the *descending* direction of the current: the latter should always be allowed to pass *continuously*, without any break or interruptions. Too great care cannot be taken that the application should be mild in character and *short* in duration; otherwise the benefit sought for would not be realized.

“Central galvanization,” in some instances, will be productive of good results, especially in gastrodynia, and more particularly when the general health of the patient is impaired. “General faradization” has sometimes been successful in my hands; for which suggestion Drs. Beard and Rockwell deserve great credit.

Faradization, locally applied in the course of the sciatic nerve, has afforded immediate and permanent relief in some recent cases of sciatica that I have treated. Nearly all practitioners are unanimous in their praises of electro-therapeutics in neuralgia.

LECTURE XL.

GLOSSO-LABIO-LARYNGEAL PARALYSIS.

Symptoms.—Difficulties of Articulation.—Paralysis of the Tongue.—Dysphagia.—Danger from Complications.—Diagnosis.—Prognosis.—Etiology.—Morbid Anatomy.—Nerves involved.—Pathology.—Dr. Clymer's Views.—Treatment.

GENTLEMEN,—You have perhaps heard of an affection called *glosso-laryngeal paralysis*. This name was applied to it by Trousseau, while Duchenne preferred *glosso-labio-laryngeal paralysis*. The distinctive character of the affection seems to have been first clearly indicated by Duchenne, who referred it to a common disease affecting the muscles of the lips, of the tongue, and of the soft palate. About twenty years prior to the publication of Duchenne's observations, Trousseau had observed a number of cases of this affection: still, it was Duchenne who first gave a systematic description of the disease. The appellation means, in plain English,—glosso, in reference to the tongue; labio, as regards the lips; laryngeal, the larynx,—a paralysis of the tongue, of the lips, and of the larynx. The disease has also been named glosso-labio-pharyngeal-laryngeal paralysis, on account of the muscles of the pharynx also becoming involved.

Like progressive locomotor ataxia, myo-sclerotic paralysis, and several other forms of nervous diseases of which I have spoken during this course of lectures, glosso-labio-laryngeal paralysis is an affection progressive in character: hence the tendency of the disease to advance and develop from bad to worse. We therefore classify it among the progressive paralytic affections.

As regards the *symptoms* of glosso-labio-laryngeal paralysis, they are very plain and conclusive, and cannot lead a careful diagnostician into error. The first symptom generally noticed is a greater or less paralysis of the lips, which paralytic condition necessarily leads to a *difficulty of articulation*, the patient pronouncing words containing vowels, especially *o* and *u*, very indis-

tinctly. Again, the difficulty embraces generally certain dental and lingual consonants. The tongue subsequently becomes involved, which, of course, causes a still greater impairment of the power of articulation.

Remember that the muscles of the tongue are never first affected,—the disease almost invariably involving the lips from the beginning. At this period, therefore, we always observe a paralysis of the orbicularis oris muscle: this produces the characteristic tendency to keep the lips apart, which is a peculiar feature of this affection, and one which you will find well portrayed in a plate in Dr. Hammond's work.

As a result of a want of tonicity in the orbicularis oris, the saliva is no longer retained in the mouth, but constantly trickles out, the patient being unable to keep the lips in close apposition. The spreading of the paralysis to some of the muscles of the tongue impedes several of its movements, the greatest difficulty at first being the moving of the tip of the tongue. You will remember that in all the forms of paralysis of which I have spoken the tongue is but rarely involved,—only exceptionally so,—and even when the paralysis is the result of a brain-lesion, we find this implication only partial and unilateral. In the disease we are now considering, the patient cannot bring the tip of the tongue to the roof of the mouth; moreover, he is unable to dislodge any portion of food remaining between the teeth and the cheeks,—not on account of a paralysis of the buccinator muscles, as is the case in Bell's palsy, but because the tongue cannot be moved in different directions. The buccinators have nothing to do with the trouble in this case; for, although part of the facial nerve becomes involved in the disease, the branches supplying the buccinators are not implicated. You will also notice, as the disease advances, that the paralysis becomes more complete, that the patient can no longer protrude his tongue, and finally that he is unable to move it at all. This organ being completely paralyzed, it assumes a guttered form, sinking in the centre, and becomes perfectly immovable. Then, in consequence of an involvement of the facial nerve in the paralysis, there is a paralysis of the velum palati and of the pillars of the fauces. This is a very serious complication, for it prevents the closing of the posterior nares during the act of swallowing, and, as a result, the alimentary matters, espe-

cially fluids, are regurgitated through the nose. Nor is this all. The alimentary bolus, not passing readily over the epiglottis, is extremely liable to lodge in the respiratory tube and produce suffocation. You see that thus the act of deglutition—an important physiological action—becomes in itself a source of danger to the patient; and the trouble may culminate in complete *dysphagia*, so that instead of a simple difficulty there may be a total inability to swallow. The act of swallowing being a source of great distress to the patient, and also of danger, he naturally shrinks from taking any food, and frequently becomes greatly debilitated. And this same difficulty, caused by the involvement of the velum palati, and later on of the pharyngeal muscles themselves, besides interfering with the introduction of food, leads to other complications. As regards the saliva, there is an especial source of danger during sleep. We have seen that, owing to a paralysis of the orbicularis oris, the saliva constantly escapes from the labial commissures, the patient preventing it from soiling his clothes by holding a handkerchief to his mouth; but during sleep it is quite a different matter, there then being danger of suffocation. The saliva, under these circumstances, has a tendency to flow towards the back part of the throat, where, under the direction of the muscles of this part, it runs down into the œsophagus. But, these muscles being paralyzed, the saliva is checked in its course, and accumulates near the larynx, so that the patient is in imminent danger of suffocation. Even should the strangulation be but partial, there is a continual liability to suffocative attacks; the patient awakes in distress, coughs violently, sleeps with difficulty, and is reduced to a most wretched condition.

I can but inadequately portray the difficulties connected with the taking of food in glosso-labio-laryngeal paralysis. In the first place, the orbicularis oris being paralyzed, the patient cannot avail himself of the aid of the lips, as we do in mastication; and his tongue being also paralyzed, he is deprived of the use of this organ to assist him in moving the food from one part of his mouth to another; then there is a ropy, stringy condition of the saliva, produced by the admixture of the buccal mucus; and, above all, there is the great difficulty that the patient often refuses to eat because he is afraid of being suffocated. His attempts at deglutition have so frequently been followed by violent attacks

of convulsive coughing, he has so repeatedly experienced the distressing sensation of suffocation, that he sometimes feels that to eat would be at the peril of his life. His sufferings may well be compared to those of Tantalus, of whom we read in mythology. Tantalus, you know, was condemned to eternal thirst and hunger. Whenever he attempted to drink of the limpid waters rising to his parched lips, the stream would recede; and his endeavors to grasp the branch laden with fruit above his head were constantly futile. In glosso-laryngeal paralysis the torments of the patient are equally excruciating.

I presume you are all familiar with the physiology of the different nerves. In this disease the hypo-glossal and the facial nerves are the principal ones affected; and when I tell you that it is the lower branches of the facial, and, consequently, the muscles of the lower zone of the face, that are involved, you will readily understand the peculiarities of expression which are always found in this affection. There is no paralysis of the orbicularis palpebrarum, as is invariably the case in Bell's palsy, the patient being perfectly able to open and shut his eyes; no paralysis of the third nerve, or of any of the branches that supply the muscles of the eye. Of course there is a paralysis of certain branches of the facial nerve, but it is only of those branches which supply the muscles of the lower zone of the face: the patient never laughs with his mouth, but "*with his eyes.*" He cannot even smile, the orbicularis oris muscle being paralyzed; but he can, to a great extent, express his emotions with his eyes, for the levator palpebræ and the orbicularis palpebrarum are not involved. But there are other characteristic phenomena present in this disease. The patient, for instance, cannot whistle, nor can he blow out a lighted candle. This, however, is due not solely to a paralysis of the orbicularis oris muscle, but also to an affection of the recurrent laryngeal nerve, a fact of which I shall speak again. This latter nerve is the motor nerve of most of the laryngeal muscles, and a paralytic condition of the rima glottidis occurs. This, as well as the want of action of the auxiliary muscles of respiration (to which the paralysis generally extends), causes the patient's inability to make sufficiently strong respiratory efforts. The progressive nature of the disease leads finally to an invasion of the muscles of the upper extremities, the intercostals and the pectorals; so that the inability to blow out

a candle does not depend, as in Bell's palsy, upon a paralysis of the buccinator, or even of the orbicularis oris, which latter is involved, but upon the paralysis of the auxiliary muscles of respiration, by which a current of air can no longer be forcibly directed towards one object, the current produced being not only weak, but broken up in all directions, part of the air escaping by the mouth, and part going through the nostrils; in short, it admits of no concentration.

You thus see that the peculiarities of this affection are quite characteristic, and so varying and different from the phenomena presented by any other form of paralysis as to be almost pathognomonic of glosso-labio-laryngeal paralysis. It is only by gross carelessness that you could mistake this for another affection. As a matter of course, as soon as the recurrent laryngeal nerve becomes affected—the patient being already unable to swallow—all power of speech is lost. Again, we know that the hypo-glossal nerve being involved, the muscles of the tongue are paralyzed, and that part of the facial nerve being affected, the orbicularis oris no longer acts; and, finally, as a result of this double involvement, the patient loses the power of articulating certain vowels, as well as labial and lingual consonants.

But what happens when the physiological function of the laryngeal muscles is suspended? There will be, of course, a complete loss of phonation,—a voicelessness,—and simply by the implication of the recurrent nerve in the disease.

Now, you can readily imagine the sad picture which is presented by an individual suffering from confirmed glosso-labio-laryngeal paralysis. As long as he is determined to make efforts at swallowing an alimentary bolus, he throws his head backwards, in order to favor gravitation towards the œsophagus. The orbicularis oris not working, there is a tendency of the food to spread outside of the teeth: this he has to counteract by placing his hand against his mouth, thus supporting the lips. Even with the head thrown backwards, however, he finds it a very dangerous manœuvre, for occasionally a small portion of food gets into the larynx, and throws him into a terrible fit of coughing, almost threatening to asphyxiate him; and liquids are quite as disagreeable as solids, for they are often forcibly ejected through the nostrils. Then, to add to his misery, he is entirely unable audibly to communicate

his thoughts to others,—the highest prerogative of a human being! Can any one fancy a more terrible condition than such a combination of ills, steadily growing worse? Should you notice such a patient during sleep, you will observe that he assumes a peculiar decubitus, and that his saliva flows out on the pillow. I have told you how the accumulation of the saliva in the posterior part of the mouth, and in the throat, may become a source of danger. In order to avoid this, and also in order to avoid being constantly aroused by these fits of spasmodic coughing, the patient throws his head forward in a peculiar way before going to sleep; which position allows the saliva to escape from the mouth by its gravitation.

Glosso-labio-laryngeal paralysis is not a disease affecting the mind; consequently, the intellectual faculties are not impaired. It is a disease involving a part of the floor of the fourth ventricle, the medulla oblongata, and certain parts at the base of the brain, as well as the particular nerves having their origin in these parts. The disease being a progressive one, it extends to and affects the auxiliary muscles of respiration, as well as the muscles of the arms; it is, of itself, a hopeless affection; and, if the sufferer is not carried away by an intercurrent disease, he dies of debility, or from accidental suffocation while eating. But a great danger is the origin of a complication of some thoracic disease, such as bronchitis or pneumonitis. The result then is necessarily fatal; for, the muscles of the thorax, as well as those of the larynx, being paralyzed, the patient is utterly prevented from making powerful expiratory efforts, such as coughing or expectoration. It is the want of power to expel the accumulating mucus by expectoration which is one of the main causes contributing to a fatal termination.

As regards the *diagnosis*, considering all I have said about the symptoms, it is hardly possible that you should mistake this for some other affection. You will certainly not mistake it for Bell's palsy, recollecting, undoubtedly, that this is a paralysis of the facial nerve *only*. Again, Bell's palsy is generally a unilateral disease, affecting the muscles of one-half of the face; it involves the occipito-frontalis, the corrugator supercilii, the orbicularis palpebrarum, the buccinator, and the facial muscles of expression. You also remember the pathognomonic symptom of Bell's palsy, which is,

that the patient cannot close his eye. This is certainly not the case in glosso-labio-laryngeal paralysis, in which disease the patient "laughs with his eyes only," and has perfect control of all the muscles of the upper part of the face. Neither can you mistake the disease for a partial paralysis of the tongue depending on a hemiplegia, since in such cases you always find certain cerebral manifestations, especially symptoms of a psychical character. It is true that one hypo-glossal nerve may be involved in a hemiplegia; but then only one side of the tongue will be affected, and the organ on being protruded will point towards the paralyzed side. Of course the set of muscles on the paralyzed side, not receiving the necessary innervation, cannot act and counteract the antagonistic set on the healthy side; and, in consequence, when the tongue is protruded it is pushed to the palsied side by the non-affected muscles. In glosso-labio-laryngeal paralysis, after the disease has lasted for a certain period the tongue cannot be protruded at all; and, moreover, as the disease advances, it cannot be moved even sufficiently for the tip to touch the roof of the mouth. So that in this case, again, there is no possibility of committing an error of diagnosis.

The *prognosis* of glosso-labio-laryngeal paralysis is extremely unfavorable, the disease being progressive, and entirely hopeless. The ordinary course of its duration is about two years. Of the *etiology* of the affection we know so little that we need not even consider the few ideas which have been advanced, and which really cannot claim our attention.

But what of the *morbid anatomy*? Where do we find lesions? Those of you who are anatomists will readily understand which are the muscles involved in a paralysis of the lips, the tongue, and the larynx, as well as of the soft palate and the fauces; you will also know the different branches of the several nerves supplying these muscles. Still, it is not alone necessary to have a knowledge of the anatomical lesions; you should also be physiologists, and understand the physiological actions of the nerves involved. And what are the nerves involved in glosso-labio-laryngeal paralysis? The hypo-glossal nerve, that part of the facial which supplies the lower zone of the face, and the part which, through the otic ganglion, innervates a portion of the soft palate; then certain branches of the pneumogastric, the recurrent laryngeal and the

pharyngeal branches first, and afterwards certain cardiac and pulmonary branches. It is when the disease is well advanced that certain physical symptoms are shown which evidently point to an impairment of the last-named branches of the pneumogastric. We find, for instance, that the pulse beats more rapidly, indicating an increase in the frequency of the heart's action; yet the respiratory process is retarded, the number of respirations being abnormally diminished. We know that one branch of the pneumogastric nerve acts as a "check-nerve" of the heart, its physiological function being to control the heart's action, and in the later stages of the disease this physiological function seems to be interfered with, the beat of the heart being so accelerated as to render it difficult to count its pulsations. Again, we know that certain branches of the pneumogastric have a decided influence upon the respiratory process; and it has been shown by physiological experiments that when the "check-nerve" of the heart is divided, the contractions of the heart increase in frequency; while, when the branches presiding over a part of the respiratory process are interfered with, the respiration is impeded and reduced in quickness, the breathing being slower than usual. We know that the hypo-glossal nerve is involved, for we have seen that the patient cannot protrude the tongue, neither can he bring its tip to the roof of the mouth. We have seen that the muscles of the lower part of the face are implicated, proving that part of the facial nerve is at fault; and we have also found that the pillars of the fauces and the muscles of the pharynx are paralyzed, proving that the pharyngeal branch of the pneumogastric is involved. Moreover, we have observed a loss of phonation, an absolute voicelessness, showing that the recurrent laryngeal and spinal accessory are also impaired. So that we have a paralysis of the hypo-glossal, part of the facial, spinal accessory, and part of the pneumogastric.

Knowing all this, the question naturally suggests itself, Where is the *site of the lesion*? It was formerly taught that the disease consisted in an atrophy of the roots of those nerves which supply the affected muscles, or of the roots connected with those fibres which are ultimately distributed to these muscles. But the science of pathology is progressive, and it is now held that not only is there an atrophy of those roots, as is evidenced

on post-mortem examination, but an atrophy also of the nuclear origin of the roots of the nerves involved; and the common deep origin of all the nerves involved in glosso-labio-laryngeal paralysis is the floor of the fourth ventricle. The disease is an atrophy of the roots; it is a pigmentary degeneration involving the nuclear origin of those cells with which the nerve-roots are connected. Dr. Meredith Clymer, of New York, who has thrown a great deal of light upon many obscure pathological questions, and of whom as an American we cannot be too proud, beautifully expresses the pathological condition of this affection when he says that it "is a pigmentary disease of an atrophic character, involving the federation of nerve-cells from which the affected nerves originate, and that these nerve-cells exist in nuclear communities, which are echeloned along the floor of the fourth ventricle, and are, by means of multiple fibres, in connection with each other." It is really impossible to find a more beautiful explanation of the morbid anatomy of this disease; it is clear and concise, and you should not allow it to escape your memory.

As for the *treatment*, you have long since learned my views about the treatment of progressive affections. Remedies, in cases so hopeless as this, are really of no avail. All you can do is to use palliative measures, at the same time not losing sight of the important fact that good hygienic conditions are necessary to counteract the great tendency to increasing debility which exists. You may also have recourse to faradization of the affected muscles, though without any prospect of aborting the paralytic affection.

And this, gentlemen, brings us to the close of the present course of lectures, but not to the end of our subject, for I might continue for six months longer, and even then I should not have exhausted the theme. The study of nervous diseases, as you are aware by this time, is a most difficult one; but still more difficult is the art of imparting to others a knowledge of these important affections. I have earnestly striven, without minuteness of detail, to clear up most of the mysteries connected with this branch, and I sincerely trust that I have so far succeeded as to enable you to pursue your studies with a full and clear understanding of all the principal features of each nervous affection which has been considered.

I have striven to give you all the latest views and discoveries so far as developed in the literature of the subject. I have had constantly to consult all the recent medical journals of importance containing articles bearing upon this department of science, as well as a great many works of different authors which to you would have been difficult of access. In this course of lectures I have laid the foundation only, and it is for you to erect the superstructure, of the edifice of scientific knowledge.

THE END.

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